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The Journal of Pediatrics

VOL. 1 DECEMBER, 1932 NO. 6

Original Communications

THE RESPONSIBILITY OF THE CHILDREN'S HOSPITAL TO ITS INTERNES

THOMAS B. COOLEY, M.D.
DETROIT, MICHIGAN

MY SUBJECT is suggested by reflection on some problems under consideration by the Committee on Education of the American Academy of Pediatrics, which is taking a part in following up the work of the corresponding committee of the White House Conference.

Medical education has become a prolonged and complicated process. It is generally understood that the curriculum of the medical schools, even the best of them, does not in itself constitute adequate training for practice, and that a certain amount of postgraduate hospital experience is a necessity before the young physician can safely be trusted to deal independently with life and health in private practice. This necessity is so obvious and so universally recognized that a number of the states now require a year's interneship before granting a license to practice. A very evident corollary to this, which I think is not usually formally recognized by hospital authorities, is that every hospital whose house staff is made up of recently graduated physicians becomes automatically a definite part of the machinery of medical education and assumes a clear responsibility to provide for these young men the best facilities in its power for the training which they need. It is responsible for this first, to the community whose needs later these men are to serve; second, to the internes themselves from whom the hospital receives arduous service in return for an implied guaranty of proper training; and third, to itself, because it wants its interne work well done, and because a reputation as a good training-ground will attract to it the better class of internes.

The chief idea in the minds of the founders of most hospitals has been, of course, to provide places for the proper care of the sick, and

Presidential address before the Section on Children's Hospitals, American Hospital Association, Detroit, Sept. 13, 1932.

I suppose that few of them, with the exception of those established as essential parts of medical schools, were planned with very clear ideas of their two other almost equally important functions—to train physicians and nurses, and to increase the sum total of medical knowledge by proper use of their opportunities. The intimate interrelationship of these three is so evident as to need no special discussion, and no hospital with any pretension to high standards can afford to disregard any one of them.

A children's hospital, and this applies equally to units for children in general hospitals of any size, belongs in the class of special hospitals, and its internes, almost without exception, will come to it for training in the special practice of pediatrics. The question of "specialties" in medicine, and proper training for their practice, has been a troublesome one. It has been not only possible, but customary for physicians with few special qualifications to set themselves up as "specialists" in one or another branch, and the public, notoriously at sea when it comes to the selection of competent practitioners, has had no authoritative guidance whatever for its choice.

"Of recent years there has been considerable evidence of intent on the part of the medical profession to remedy this condition. National associations in several special branches have established machinery for examination and certification of qualified specialists, and something similar may be expected before long for all of the specialties. In pediatrics up to now the only step taken has been the statement by the Academy of Pediatrics of qualifications for its membership which, while satisfactory because wisely interpreted, are not very definite. The Academy committee of which I have spoken has assumed as part of its duties an attempt to determine by collating opinions from a large number of established pediatricians what should be considered a minimum postgraduate training adequate for special practice. Recently the committee has been asked to collaborate with committees from other national organizations in a study of means to provide for examination and authoritative national certification and listing of qualified pediatricians.

Standards arrived at in this way will be presumably for a minimum amount of definite kinds of experience, both during and after the interne period, and an examination to determine the efficiency of this training. While the hospitals should be concerned with optimal training of their internes, they must be interested in minimal standards likely to set by any authoritative body which may affect their present organizations, as they must of course fall in line with such requirements. The replies which the Committee has received to its questionnaire would indicate the probability of a requirement of two years interneship in a recognized children's hospital, with some possible

substitutions such as six months in a hospital for infectious disease. Two years is longer than the average interneship in the children's hospital. If we should have to make this our minimum it would mean a very considerable readjustment for some of us. It would also mean a lessening of the number of internes graduated in a given time, and a corresponding cut in the number of available interneships. A question would arise immediately as to whether if such a requirement were put into effect the hospitals of the country could train enough men to meet a demand which up to the time of the present economic upheaval seemed to be growing steadily. It seems probable now that what we have been going through will result in some permanent changes in conditions of practice, the exact character of which we cannot yet foresee, so that this question can be answered only by experience. Another probable minimal requirement which should be of service to the hospitals would be that of a definite period of attendance in the out-patient department of a children's hospital. If every aspirant for certification as a pediatrician were required to present a certificate of satisfactory service of this character, the hospitals should have a steady supply of men who would feel obliged to give us something better than the irregular and perfunctory service with which we so often find ourselves compelled to be content.

Pediatrics has broadened its scope greatly since the time not many years ago when it was quite adequately defined as comprising infant feeding and the diseases of infancy and childhood. From that point it has grown rather rapidly but logically to arrogate to itself the supervision of all that pertains to physical and mental health and growth during the age period between birth and the end of adolescence. To cover such a field requires a knowledge of the details of normal growth and development and the variations therefrom; of the beginnings of disease and its prophylaxis; of the mental hygiene and the normal and abnormal psychology of children and their parents; and in pediatrics more than in almost any other field, a considerable experience in the difficult art of dealing with all kinds of people.

The conventional hospital set-up is not especially adapted to training in all these things, yet it is here that the interne should get his grounding in them. I think that we are at our best in the diagnosis and treatment of serious disease, and have least to offer ordinarily in opportunities to observe the normal, the beginnings of disease, and the handling of minor illness. These are obviously things to be provided for in the out-patient department. This is notoriously the most difficult department of the hospital to organize and keep running smoothly and efficiently. Hospitals differ in their visiting staff assignments for this service. I suppose the common arrangement is to leave all but certain special clinics to the younger and less experienced

members of the staff, though in a few hospitals senior staff men give service here as regularly as in the house. This is certainly the desirable plan from every point of view, and particularly with regard to what the interne is to get out of his experience there, and it seems that it should be possible if its desirability is sufficiently realized.

The out-patient department usually has a general clinic and a number of special clinics—heart, skin metabolic diseases, allergy, etc., depending on the size and character of its clientele. Certain special clinics not found in all hospitals should, I think, always be arranged for, if for no other reason than that they are the best places for the internes to get certain kinds of experience. One of these is the "well-baby" or "prophylactic" clinic. This is often a poor thing, whose activities are devoted mainly to weighing babies, ordering milk mixtures and cod liver oil, and injecting toxoid. A large part of the living of the modern pediatrician is earned by the supervision and prophylactic care of well children, and a clinic of this kind, arranged and supervised by a man really interested in normal growth and development, could be one of the most valuable of the interne's experiences. It seems to me that there is a possibility, by cooperation among those interested, of attaining some kind of standardization of these clinics on a higher plane than most of them reach at present.

The question of what to do about mental hygiene and "child guidance" or "habit" clinics is a vexing one, and one that has been much to the fore recently. Unfortunately, it has been sadly befogged by faddism, so much so that most of us have not a very clear idea of just how the experienced pediatrician, the psychiatrist and the lay "child psychologist" are going to fit into its ultimate solution. Certainly, so far as anything like a standard method of dealing with it is concerned, it is still in the experimental stage. The children are there, however, to be dealt with, the internes (and the staff) need experience in their handling, and probably the individual hospital will contribute to the working out of the problem by establishing its clinic according to what seems to it the method most available in its particular circumstances

Assuming that the out-patient department has the ordinary number of special subdivisions, the internes should rotate through these throughout their term of service. They should get more real benefit from this type of experience in the latter part of their term than earlier. They should not, however, have their other work interfered with by constant calls to substitute for some attending physician who has failed to meet his appointments. I do not suppose that ours is the only hospital where this is a frequent happening.

Just how much the interne gets out of his service on the wards, aside from the opportunity to see and care for patients himself, must

depend of course greatly on the character and attitude of the visiting physicians. Some men are born teachers: some who are not might be better if they tried, or if they realized a distinct obligation to teach what they can. Ward rounds are often not properly arranged for teaching because they come at such irregular times that the internes cannot so plan their work as to be sure of being able to follow them. The man who starts his rounds at ten o'clock on one day and at twelve the next is not very considerate of his internes' rights and is seldom much of an asset to the hospital. Some things of great instructive value to both internes and staff members are not well planned and carried out in all hospitals. The clinical-pathological conference, for instance, is an especially valuable institution, in which it is sometimes surprisingly hard to get regular participation by staff members who might help to make it successful and benefit themselves at the same time. Meetings between internes and staff members for discussion of case histories and records benefit the internes and the hospital more than the staff members, perhaps, but are highly desirable.

It would be easy to go on enumerating details of this kind, the main point of which is to illustrate our obligation to plan the hospital arrangements so that the interne may get the training he needs and is entitled to.

A certain number of young men anxious to get a better training than they can acquire in the standard two years will want a three years' service. The ordinary method of arranging this is to take a third year as resident physician. When the man knows at the beginning of his service that he will want three years, it would be possible to plan a better arrangement, which might include a year's work in the laboratories, pathological, chemical or both. The interne system might well be made flexible enough to allow for such services for the more promising candidates. There is a real advantage to the interne and to the hospitals in arrangements by which men taking the longer training divide their time between hospitals in different parts of the country. Such interchange goes on now to some extent. I expect to see it become more common, and perhaps more definitely provided for as we take more thought to our system of interne training. Some hospitals are so fortunate as to have fellowships at their disposal. The complete development of the most capable of our young physicians is an important service to the community, and hospitals blessed with generous patrons might well encourage such foundations, which would be of advantage to themselves as well as to the physicians and the community.

All that I have been saying can be summed up very briefly. We have a definite responsibility to our internes, of which many of us have not been sufficiently conscious, and our plans to discharge this

responsibility should be as careful and as systematically carried out as those for any other function of the hospital. The chief burden of this must fall, of course, on the professional staff. Whenever one contemplates the constantly increasing complexity of medical service in public or semipublic hospitals, such as most children's hospitals are, one must, I think, be impressed with the idea, becoming constantly stronger among the medical men, that the traditional system of wholly voluntary medical service is archaic, and no longer adequate for the needs and responsibilities of the modern hospital of any size. A certain amount of voluntary service is and always will be proper, because the attending men receive a real compensation in the form of valuable experience; but adequate supervision and correlation of the manifold activities of an up-to-date hospital requires an amount of time and thought on the part of some one at least of the medical staff that he cannot possibly afford to give without some remuneration. This is a poor subject to contemplate at a time when budgets are so difficult to meet as just now, but there is no use in blinding ourselves to the fact that there must be some change in hospital policy in this respect if we are to do our work as well as the community expects us to.

7815 Jefferson Avenue, East.

CALCINOSIS AND SCLERODERMA: TREATMENT OF A CASE BY USE OF THE KETOGENIC DIET

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ROCHESTER, MINN.

ACCORDING to Basch, attention was first directed by G. Thibierge and R. J. Weissenbach to the anatomicoclinical syndrome of calcareous concretions in the skin and subcutaneous tissues in scleroderma. Earlier reports by other authors made only passing mention of the coexistence of the two phenomena and indicated that it was but a fortuitous coincidence. Basch pointed out that the syndrome is relatively rare and that she had been able to collect reports of only forty-five cases from the literature. Her monograph presents a complete résumé of the various aspects of the syndrome, which she designated that of Thibierge-Weissenbach.

The condition may be in circumscribed or diffuse form, and of each form there are two types. The first type of the diffuse form affects women almost entirely (twenty-six of twenty-seven patients in reports found by Basch) and occurs, as a rule, in the course of Raynaud's disease and thyro-ovarian upsets. It is therefore apparent that in this type vasomotor disturbances, acrocyanosis, paresthesia, angiospasm, local syncope, and pain will be found early in the course of the disease. The features of this type are the onset as a sclerodactylia, calcareous concretions with characteristic subcutaneous situation in the tissues of the digits; relative rarity of involvement of the muscles and skeleton, and slow evolution with relatively favorable prognosis.

Of the second type of the diffuse form Basch was able to collect eleven reports from the literature. As with the first type it has a predilection for females (nine of eleven cases) but differs from the first type materially in that it is an affliction of the young, occurring mainly between the ages of eight and fifteen years. This juvenile type, designated by Basch as chronic edematous scleroderma with "sclerotendonitis" and "scleromyositis," exhibits calcification, which localizes characteristically in the proximal portion of the extremities and in the trunk, which is abundant, and which occurs superficially and deeply. The extent of the muscular lesions and the general debility make the outlook in this type very grave. The immediate causes of death in three cases were progressive cachexia with secondary infection, staphylococcic infection, and tuberculosis. In one case improvement followed elimination (extrusion) of the calcareous masses. The other cases were not followed.

From Section on Pediatrics, The Mayo Foundation.
Submitted for publication, July 12, 1932.

In addition to the two types of the foregoing form Basch recognized two types of the circumscribed form: the calcareous concretions of scleroderma "en plaques" ("morphee en plaques"), and the other, calcareous concretions of scleroderma "en bandes." It is obvious that the circumscribed form is milder than the diffuse form, for in the former the calcareous masses are localized.

Basch considered the numerous hypotheses which have been advanced in explanation of the pathogenesis of the condition. There are two chief lines of thought: first, that there is a constitutional condition which favors precipitation of calcareous salts in the tissues, and second, that local changes in the connective tissues result in increased affinity on the part of the tissue for calcium salts. These explanations are supported and contradicted by numerous arguments based on histology, anatomy, and pathology.

Little can be said for the results of the various forms of treatment which Basch mentioned. Spontaneous resorption of calcareous deposits is possible, and there may also be spontaneous extrusion of some such deposits through the integument. In cases of scleroderma and calcareous deposits in conjunction with Raynaud's disease, treatment of the Raynaud syndrome by the newer methods has resulted in favorable influence on calcification. Amelioration may follow a regimen combining hygienic measures with one of a number of medicaments such as acetylcholine, insulin, salts of pilocarpine, iodides, especially organic preparations in which iodides are combined with thiosinamine, phosphoric acid, organotherapy, physiotherapy, and finally surgery. It is of particular interest to consider these measures in reference to the type of treatment adopted in the case reported here, especially in view of the fact that marked changes in the extent of calcareous deposit has not been reported formerly.

In the American literature on this subject, Durham's contribution represents a full review of the various features of scleroderma and calcinosis, and his consideration, together with Basch's cover the subject quite fully. Previously, I reported the present case in part. My purpose at that time was to record the case because of its rarity, and also because of the wide extent of calcareous deposits. As was stated at the time, the patient had been receiving a ketogenic diet for three and a half months. Her course up to that time was reported, but observation as a basis for the report was by the parents, not by me or another physician. Since the first report, the course taken by the process has been such as to justify an abstract of the previous report and bringing of the history up to the present time (June, 1932).

REPORT OF CASE

A girl, aged six years, was first brought to the clinic in September, 1928, because of masses in the skin of the elbows, axillae, and back, and because of difficulty in walking. She had had frequent attacks of vomiting until the age of five years.

In September, 1926, instability and difficulty in walking had been noted, and various methods of treatment had been without much avail. At the time of examination, September, 1928, braces were worn half of the day. If the braces were left off for a period of two weeks, there seemed to be a tendency for the return of contracture at the knees. In the axillae, and antecubital and popliteal spaces, over

Fig. 1.—September, 1928. Diffuse extensive calcareous deposits in the subcutaneous tissues about the lower part of the spinal column and hips. Calcareous masses can also be seen in the tissues of the axillae.

the lower part of the spine, and surrounding the region of the pelvis were firm, bony-hard deposits apparently in the subcutaneous tissue. The skin in these regions was smooth, rather shiny, and firm. There was marked limitation of motion in most of the joints, especially the hips, knees, elbows, and shoulders; the gait was peculiar and stiff-legged. The value for serum calcium was 10.8 mg. for each 100 c.c. (normal, 10 mg. for each 100 c.c.); the value for serum phosphorus was

4.2 mg. for each 100 c.c. (normal, 5 mg. for each 100 c.c.). Roentgenograms of the trunk (Fig. 1) and extremities showed calcification of the skin and subcutaneous tissue around the lower part of the pelvis and in the arms, forearms, axillae, and around the knees. The roentgenogram suggested the diagnosis of scleroderma. There was no roentgenologic evidence of disease of the joints or bones.

The child was placed on the ketogenic diet, as described by Helmholz, and at the end of the first week was receiving carbohydrate 14 gm., protein 20 gm., and fat 140 gm., and was permitted to return home. Her weight at this time was 39 pounds, a loss of 3 pounds since the commencement of the diet. This loss of weight occurs in nearly all children during the first two or three weeks on the diet.

One and a half months later the mother reported that the child was walking without braces. Diminution in the size of the masses in the skin was not ap-

A. *B.*

Fig. 2.—*A*, September, 1929. Diffuse deposits of calcium about pelvis and thighs. Compared with Fig. 1, some decrease in the amount of calcareous material present in the region of the hips is noticeable; *B*, calcareous material in subcutaneous tissues of the thigh. The distribution in the fascial sheaths is evident.

parent although they seemed somewhat softer. Two and a half months after dismissal the child had gained 1.5 pounds after the diet had been changed to 14 gm. carbohydrate, 25 gm. protein, and 155 gm. fat. Three and a half months after dismissal the weight was 43.5 pounds. The stiffness which had formerly appeared after sitting had disappeared and the child was walking better. Her general condition was better and her color was good. However, all of these data indicating improvement had been reported before, and were obtained from the parents. New data can now be given.

The child returned to the clinic in September, 1929. At that time the mother stated that the child's condition was very markedly improved. She had noticed

that when the child received a bruise or a scratch, a calcareous deposit appeared at the site of injury. The gait was markedly improved, although there was still some stiffness. The calcareous masses were still palpable in the antecubital spaces and about the hips, thighs, and knees. The urine test was positive for acetone and diacetic acid. The value for serum calcium was 10.9 mg. in each 100 c.c. and for inorganic serum phosphorus, 4.5 gm. in each 100 c.c. Roentgenograms gave evidence of extensive calcification of the soft tissues about the lower part of the spinal column, the hips, pelvis, and both thighs (Fig. 2 *A* and *B*). There was also considerable calcareous material in the axilla (Fig. 3). The weight at this time was 52 pounds, which was the normal weight for the patient's height and age. The diet was continued until January 1, 1932. It was discontinued at this time partly because the child was tiring of the restrictions imposed and partly because the mother reported that the child had apparently recovered.

The child returned to the clinic in June, 1932. At that time she weighed 79.5

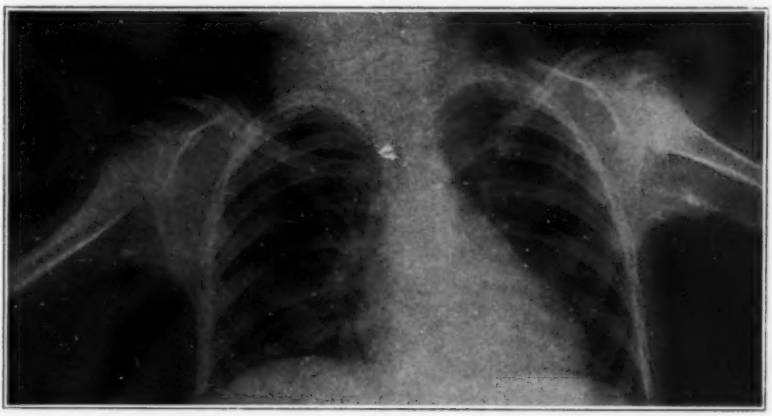
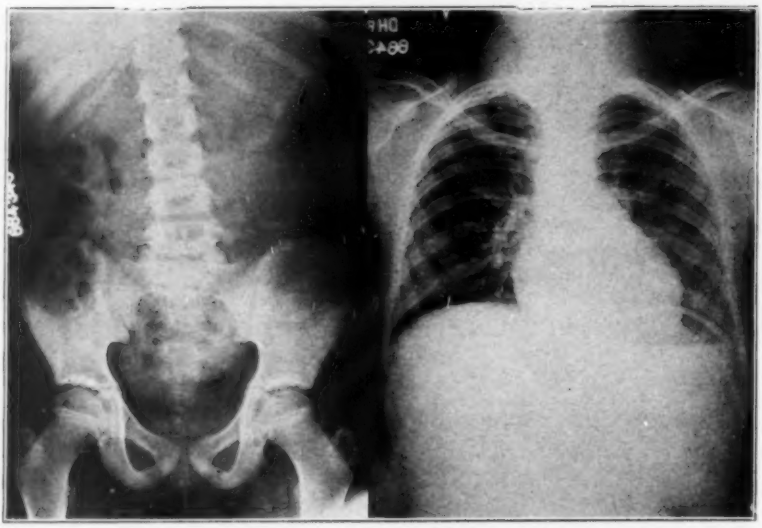


Fig. 3.—September, 1929. Considerable calcareous material in axillae.

pounds as opposed to a normal weight for her height and age of 70 pounds. There was genu valgum, with slight awkwardness of the gait and pronation of the feet. There was no demonstrable limitation of motion of the joints. The condition of the teeth was pronounced normal. Two masses only were palpable, one on the inner aspect of the right knee and one on the inner aspect of the upper part of the left arm. The color and the texture of the skin over the affected portions were normal. There was some subcutaneous atrophy or fibrosis, which resulted in a feeling of firmness. The value for serum calcium was 11.4 mg. in each 100 c.c. The value for blood phosphorus was 5.1 mg. in each 100 c.c. Examination of the urine gave negative results. The concentration of hemoglobin was 80 per cent. Erythrocytes numbered 4,290,000 and leucocytes, 4,700 in each cubic millimeter of blood. Roentgenograms of the pelvis, spinal column, and axillae were negative (Fig. 4 *A* and *B*), the calcareous deposits having entirely disappeared. Roentgenograms of the knees gave evidence of a number of small regions of calcification. There was also slight increase of calcium in the proximal end of the diaphysis of both tibias. The child was permitted to return home with a diet from which only milk was excluded.



A. *B.*

Fig. 4.—*A*, June, 1932. Calcium in the region of hips, pelvis, and lower part of spinal column has disappeared; *B*, calcium has disappeared from the soft tissues of the axillae.

COMMENT

Unfortunately metabolic studies could not be made in this case. Values for serum calcium and phosphorus were determined on three occasions: first, in September, 1928, at the time of the first visit to the clinic, and again in September, 1929, and June, 1932, at the time of subsequent visits. In every instance the values were normal. These are of little significance without knowledge of the intake and output of calcium and phosphorus, as well as of other metabolic factors. Although there is no evidence indicating abnormal calcium or phosphorus content of the osseous system, in the roentgenograms made in 1929 and in 1932 there is a definite transverse line indicative of disturbed ossification at some previous time, and it is fair to assume that such a disturbance took place in the first two years of the disease.

It is obviously unsound to insist that this single case represents a successful therapeutic result due entirely to the use of a ketogenic diet. It is significant, however, that improvement was noticeable within six weeks after the diet was instituted, although the course previously had been increasingly unfavorable. It is further of significance that no record of a case exhibiting a similar clearing of extensive calcareous deposits has been found.

Knowledge is unsatisfactory concerning the etiology and pathogenesis of the condition. It is therefore not possible to state that any method of treatment will remove the cause of the trouble. However, a treat-

ment which apparently will produce such results as were obtained in this case is worthy of use. Its employment was based chiefly on the observations of Frölich and of Nelson. The former postulated that since an "acidotic metabolism" hinders healing of fractures and deposition of calcium in newly forming bone, it should be of help in decalcifying undesirable deposits of lime salts in the body tissues. He used an "acidotic" diet in a case of myositis ossificans with good results. Nelson found that a ketogenic diet produced a negative calcium balance in a small group of children observed over a relatively short period. Certainly, in the present case it appears that the high content of fat resulted in withdrawal of calcareous material from the sites of deposit and excretion of this material through the intestine or kidneys, or through both. That similar results can be obtained in other similar cases can be determined only by further trial.

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MASSIVE GENERALIZED TUBERCULOUS INFECTION IN CHILDREN, WITH RECOVERY

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IT IS only within the last few years that we have learned to be hopeful in our prognosis of tuberculous disease in infants and children. A tuberculous infection, especially when involvement of the lungs was present, formerly was given a poor prognosis, but we now know that most children handle moderate amounts of infection, if continuous reinfection is prevented. There is even some evidence that children take care of tuberculous disease better than adults.

Several factors influence the progress of tuberculous disease. Important among them is the number of tubercle bacilli ingested or inhaled, either at the time of the initial infection, or after allergy has been established. Another factor is the virulence of the particular strain of tubercle bacillus. Still another and important one is the individual resistance to the infection, whether it be racial or otherwise. Finally, the outcome of tuberculous disease depends upon the site of the initial infection, and to a great extent upon whether it spreads from local disease to become a generalized infection.

We are accustomed to give a less favorable prognosis, if the tuberculous disease becomes generalized, than when it remains localized. There is also a belief that if generalized tuberculous disease occurs in an infant or a young child the outcome is usually fatal.

The following three cases are presented as examples of the healing of rather extensive generalized tuberculous infection in children, where it may sometimes heal by absorption, and sometimes by calcification.

CASE REPORTS

CASE 1.—K. L., Chinese boy, aged fifteen months. Entered the hospital with the complaint of cough of three months' duration, loss of weight over one month, and distention of the abdomen for two weeks. His birth weight was nine and a half pounds. The patient had always been well up to the time of the present illness, which began with the symptoms of a cold, followed very soon by the cough, and later by chills and fever. The appetite began gradually to fail, with a failure to gain in weight, followed by a rather rapid weight loss. Signs of prostration developed, and he vomited frequently after feedings. About one month before entry the abdomen began to swell and had been distended ever since, with an ac-

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Read before the Pediatric Section of the California Medical Association, at the Sixty-first Annual Session, at Pasadena, California, May 2-5, 1932.

companying diarrhea. The father had active pulmonary tuberculosis, and tuberculosis of the knee. His cough was accompanied by much expectoration. The mother, father, and four children all lived together in a single room. Physical examination showed an extremely emaciated infant weighing sixteen and three-quarters pounds. The head was large, with parietal bossing. Fine râles were present over both lungs. A rachitic rosary was present. The abdomen was protuberant and bloated. The neck was stiff, and held rigidly when an endeavor was made to flex it. Over the skin of the body were numerous small indurated and slightly ulcerous lesions, scrapings from several of which showed acid-fast bacilli. These latter lesions were taken to be tuberculides. The blood and urine were

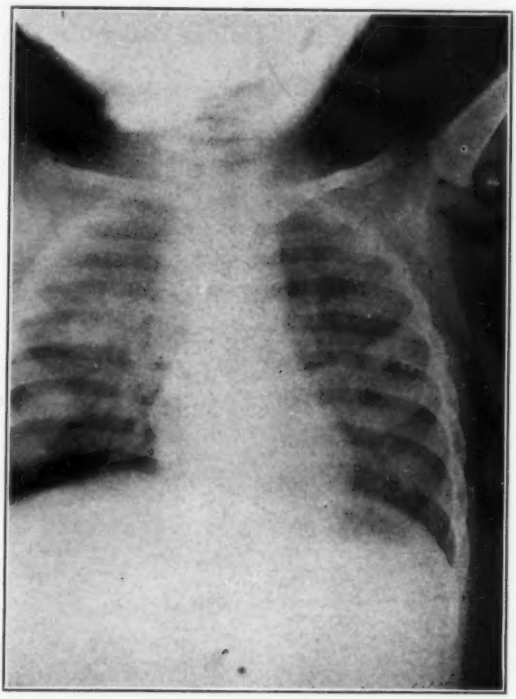

Fig. 1.—Case 1, showing the massive involvement of the right lung field.

normal, and the Wassermann was negative. The intracutaneous tuberculin test was positive. Spinal puncture showed a clear fluid under considerable pressure, with ten lymphocytes per c.mm., and only a trace of sugar. No tubercle bacilli were found. Roentgenographic studies showed generalized infection of the lung fields, with massive involvement on the right side (Fig. 1). While in the hospital the patient ran a daily fever, sometimes as high as 103° F., for more than two months. The diarrhea continued for some time, but gradually subsided. The fever resolved and from three months after entry until the time of dismissal, one year later, was normal most of the time. Heliotherapy was applied after the afebrile period had begun. The child's appetite returned, he began to gain in weight, was entirely well and negative on physical examination at the time of dismissal, with

a weight of thirty-two pounds. Roentgenograms taken at various times during his stay showed a resolution of the chest infection, which was gradual at first, but seemingly complete on dismissal.

CASE 2.—L. C., Chinese boy, aged eight years. The presenting complaint was pain in the left heel, of two months' duration. About eight months previous to entry swollen glands in the neck and inguinal region had been noticed, and these had increased gradually in size. The boy was said to have lost about fifteen pounds in the previous eight months. At the age of three an adenotonsillectomy had been done. The family history was entirely negative. Physical examination revealed a fairly well developed but poorly nourished boy. There was a large swelling at the

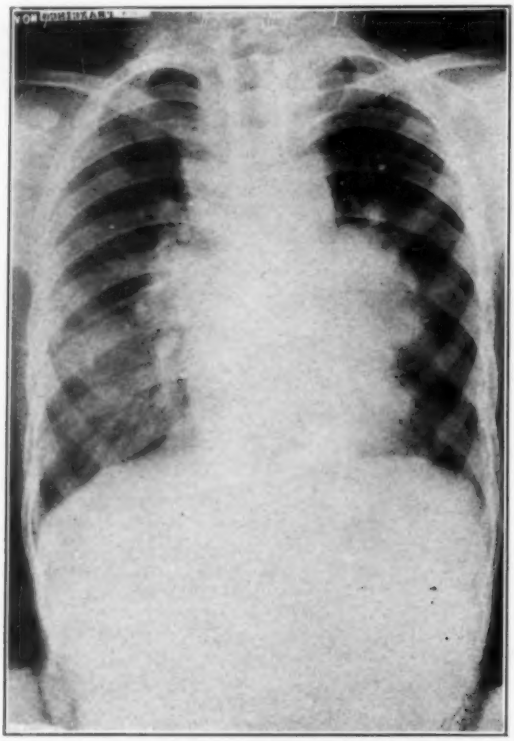

Fig. 2.—Case 2. Ventral projection of chest, showing large masses at lung roots.

angle of each jaw, which were obviously lymph nodes. There was in addition a marked generalized palpable lymphadenopathy which included the cervical, submental, supraclavicular, axillary, epitrochlear, inguinal, and popliteal lymph nodes. There was a soft nodule about almond-size in the right side of the soft palate. On the right fourth rib, near the midaxillary line, was a hard nodule about the same size. There was definite parasternal and paravertebral dullness on percussion. The blood showed 4,000,000 red cells, 70 per cent Hb. (S), 5,000 white cells, with 55 per cent polymorphonuclears, 19 lymphocytes, 13 large mononuclears, 5 eosinophiles, and 8 basophiles. The urine was normal, and Bence-Jones protein was absent. The Wassermann was negative. The tuberculin was strongly positive. Bi-

opsies were done on a right inguinal node, the left epitrochlear, and on the nodule over the right fourth rib, all of which proved to be typically tuberculous lymph nodes. Part of the specimen from the arm was ground with sterile sand and normal salt solution and filtered through sterile cotton. Petroff's medium showed a growth of acid-fast bacilli. Two guinea pigs, three young chickens, and two rabbits were inoculated, tuberculosis being produced only in the guinea pigs. Roentgenographic studies were made of the chest and bony skeleton. Examination of the chest demonstrated large mediastinal masses surrounding the trachea and major bronchial branches (Fig. 2). Aside from the rather widespread pleural thickening, there was nothing in the lung fields to suggest infection. In the skeleton

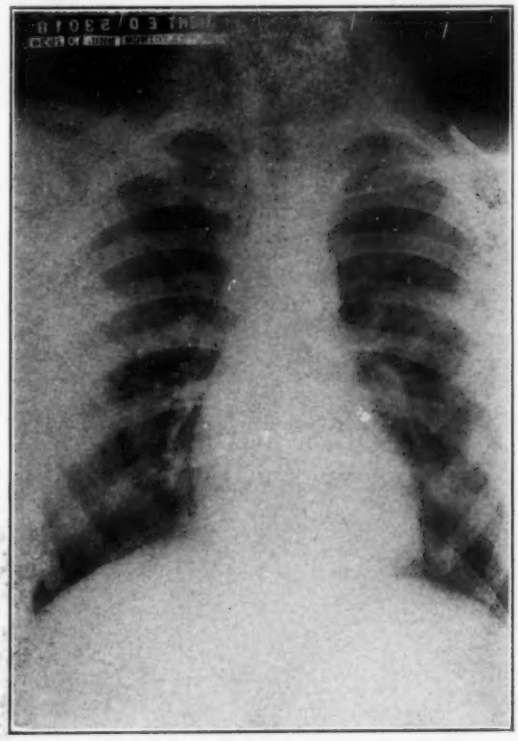

Fig. 3.—Case 2. Same projection as Fig. 2, taken two years later, showing disappearance of the lung root masses.

there were shown multiple areas of bone destruction. These lesions occurred in the pelvis, skull (Fig. 4), each os calcis, and in the ribs. A well-defined, somewhat irregularly rounded area 2½ cm. in diameter appeared in each innominate just above the acetabulum and in the left innominate a smaller area of necrosis showed near the sacrum (Fig. 6). Each heel demonstrated a poorly defined region of necrosis just above the epiphyseal scale (Fig. 8), and two ribs showed areas of involvement (Fig. 2). Four months after the admission of the patient to the ward, a fluctuant swelling suddenly appeared over the sternum. Roentgenographic examination of this revealed a large sternal lesion, a portion of the body of the sternum being destroyed with accumulation of a soft tissue swelling and calcified débris

(Fig. 10). During the first two months of the patient's stay on the ward his weight fell from fifty-three to forty-nine pounds. He was started on rather intensive heliotherapy, and began a rapid and steady gain in weight. Two years later, at the time of his dismissal, he was entirely symptomless subjectively, and

Fig. 4.—Case 2. Left lateral view of skull showing one centimeter area of necrosis in left parietal bone near the lambdoid suture.

Fig. 5.—Case 2. Same view as Fig. 4, two years later, showing normal appearance of skull.

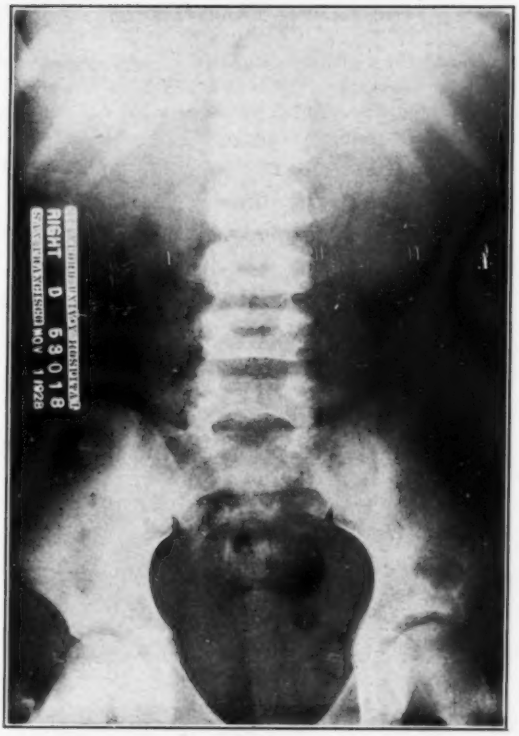

Fig. 6.—Case 2. Pelvis, demonstrating the large irregular areas of necrosis in each iliac bone suggesting metastatic tumor in appearance.

Fig. 7.—Case 2. Same view as Fig. 6, two years later, showing slight alteration in the pattern of the trabeculations to mark the site of the old necrotic areas.

weighed ninety-two and three quarters pounds. All of the swellings and lymph node enlargements had disappeared. Chest films taken at this time revealed surprising disappearance of the mediastinal masses, with only the slightest lung root calcification and pleural thickening remaining (Fig. 3). Lateral film indicated two or three large glands at the tracheal bifurcation. The lesions in the skull (Fig. 5) and in the pelvis (Fig. 7) had completely filled in with new bone, slight bone

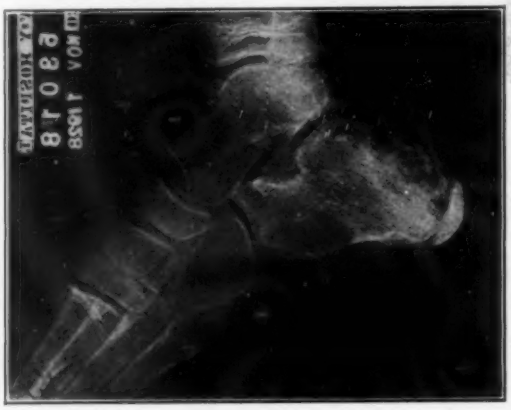

Fig. 8.—Case 2. Left os calcis, showing bone destruction in the posterior superior portion.

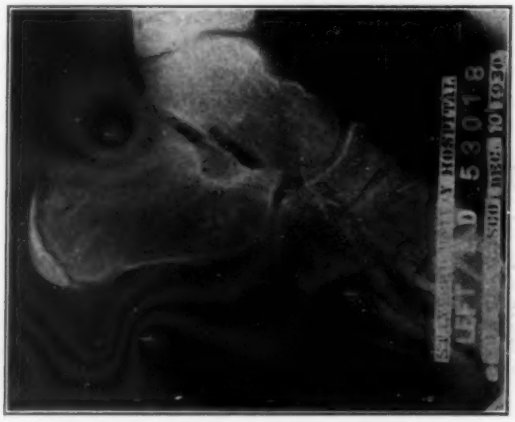

Fig. 9.—Case 2. Same view as Fig. 8, two years later. Left os calcis practically normal.

sclerosis being noted in the ilia to mark the site of the old necrotic areas. Films of the os calcis (Fig. 9) showed restoration of normal structure. At the present time, three and one-half years after the beginning of the illness, his weight is one hundred and twelve pounds. He seems entirely well, except for occasional pain in the left heel, with some limp at the time. The roentgenographs of the heel are still normal.

Fig. 10.—Case 2. Lateral view of sternum.

Fig. 11.—Case 3. Right wrist with left for comparison. The bone destruction in the ulna proximal to the epiphysis with periosteal reaction and soft tissue swelling.

CASE 3.—R. B., American boy, aged nine years. Patient reported to the outpatient clinic because of swelling of the right wrist, following immediately on an injury two weeks previously. Examination revealed a fluctuating swelling on the internal distal end of the ulna. The swelling was aspirated of 3 c.c. of turbid, yellowish fluid, smears of which showed a few acid-fast bacilli. A guinea pig was injected, killed within a month, and acid-fast bacilli were found in smears from the spleen. The Wassermann was negative. Roentgenologic study showed a sharply margined oval area of bone destruction at the ulnar side of the distal end of the diaphysis of the right ulna (Fig. 11). The condition was treated by further aspiration, followed by immobilization in plaster. Four months later the boy reported with a complaint of pain in the back when walking or standing, duration five days. On examination there was a long rounded kyphosis over the middle of the

Fig. 12.—Case 3. Lateral thoracic spine, showing destruction, in part, of sixth and seventh vertebrae.

thoracic spine. The patient could not straighten up, and there was a slight list to the right. All motions of the spine were greatly restricted, and psoas spasm was present. The patient was well developed and above the average in weight. Roentgenographic study of the spine showed a spondylitis affecting the sixth and seventh dorsal bodies (Fig. 12), with a paravertebral abscess. Films of the chest showed definite involvement with abnormal density in the left upper lobe. Hospitalization revealed a daily afternoon fever as high as 101° F. A Hibbs operation was performed by Dr. Leonard W. Ely on the thoracic spine from the fourth to the eleventh dorsal vertebra. A normal convalescence from the operation took place, and the boy gained seven pounds in four months. At the end of this time he began complaining of pain in the right hip, had a return of his fever, and extension was limited in both hips. At this time the tuberculin test was positive, films of the hips were normal, while those of the spine showed slightly increased destruction of

the sixth and seventh thoracic vertebrae, with a nonunion of several of the spines. A revision of the Hibbs operation was performed. Normal convalescence ensued, and the patient was entirely well for over two years. Eighteen months after the last operation the area of destruction in the ulna appeared in films as a sharp, smooth defect which gave no impression of activity, and there had been no progression of the deformity in the dorsal spine. The chest films meanwhile showed progressive disappearance of the mottled density with replacement by evident scarring and calcification along the bronchial tree distribution and decrease in the lung root density. The return of pain in his back, with some fever led to the discovery of a focus in the second and third lumbar vertebrae (Fig. 13); the films also revealed numerous small calcifications in the liver and spleen, interpreted as healed tubercles. An Albee operation on the lumbar spine was followed by an

Fig. 13.—Case 3. Laterial lumbar spine demonstrating spondylitis of second and third vertebrae. Calcified tubercles in liver and spleen may also be seen.

uneventful period, except that the boy had lost considerable weight which he did not seem able to regain. With the beginning of intensive heliotherapy his appetite improved, and he began a rapid gain in weight, rising from seventy-four pounds to one hundred and nine in eight months. He was dismissed from the ward one year after the Albee bone graft, entirely symptomless. At the present time, seven years after he was first seen, and aged sixteen, he is entirely well except for marked varicosities in his lower limbs.

DISCUSSION

The term "miliary tuberculosis" is not applicable to the cases we have presented, and should be reserved for those presenting numerous small lesions of uniform size, distribution, and progression, sometimes

confined to the pulmonary tissue, but often involving in addition abdominal viscera, meninges, central nervous system, bones, and joints. This type of disease is almost always fatal, although cases of recovery have been reported by Baer,[1] Blaine,[2] and others. When recovery has been reported, the healing process has usually been by means of calcification. Possibly the first case in this series could be called miliary tuberculosis because of the manifestations in the skin, but healing here and in the lungs evidently took place chiefly by absorption.

A patient similar to our second case was seen and the case reported by Pierson,[3] from the Stanford Medical School in 1926. This patient, a fifteen-year-old boy, had an involvement of the entire lymphatic system with lesions in the lungs, all healing by absorption. Our case in addition showed marked involvement of the bony skeleton. The diagnosis of the original lesions from the roentgenographic appearance presented a difficult problem. The areas of destruction in the pelvis and skull strongly suggested metastatic tumor or myeloma, and in connection with the large mediastinal masses indicated a diagnosis of lymphoblastoma. However, Assman[4] in his discussion of childhood and lymph gland tuberculosis, mentions the rare occurrence of bone lesions in combination with large mediastinal glands. Rosenblatt[5] describes an autopsied case in a male of twenty-eight years who developed metastatic involvement of the ribs, claviele, and frontal bone secondary to chronic pulmonary tuberculosis. In his review of the literature prior to 1923 he found no similar cases. Another case was described by Major and Guard[6] under the title of "Osteitis Tuberculosa Multiplex," the bone lesions of the pelvis and ribs strongly suggesting multiple myeloma. The lesion in our case in the os calcis is remarkable chiefly as evidence of the wide dissemination of the disease, since lesions of a similar nature have been described.[7]

In the third case of the present series, the successive appearance of multiple bone foci suggest metastatic phenomena, possibly from the probable primary focus in the chest. The calcified nodules in the spleen and liver indicate that at some time considerable numbers of tubercle bacilli must have been thrown into the blood stream. The processes of healing in the multiple foci were not chiefly by absorption, but rather by calcification, the usual method when the healing in tuberculous disease is complete.

How may the factors in the healing of these massive infections be explained? Probably all of those mentioned previously have entered into the healing of the disease in each case. In the first case a factor of primary importance must have been the removal of the patient from his contact, which stopped the continual reinfection. In the last two cases marked and rapid improvement began immediately after the be-

ginning of heliotherapy, indicating a probable change of the individual immunity or of the resistance to tuberculosis.

These three patients were among the first seventy children admitted to the ward for tuberculous male children at the San Francisco Hospital, indicating that recovery from rather massive infection with the tubercle bacillus in very young patients is not only possible, but probably more common than ordinarily supposed, if proper methods of care be instituted.

CONCLUSIONS

1. Three cases in male children of heavy, generalized, tuberculous infection are reported with apparent complete recovery in each.
2. Primary factors in the recovery of such patients are removal from the contact, and the instituting of general hygienic measures usually applied in the treatment of juvenile tuberculosis.
3. The disease may heal by absorption or by calcification.
4. The cases presented constitute over 4 per cent of the patients admitted to a ward for tuberculous patients.

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SOME FACTS ABOUT THE SO-CALLED NEUROPATHIC CONSTITUTION

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IN A PREVIOUS paper appearing in this Journal,* I attempted to outline a few principles involved in a practical approach to mental health problems of childhood which come to the physician in practice. The mind of child and adolescent is not, as we are apt to suppose, synonymous with the intelligence. Mind is the human being in action. Hygiene must concern itself with every factor of growth and development concerned in the total welfare of the individual. Mental hygiene is an aspect of hygiene that takes a genetic interest in the assets and liabilities of a growing organism, and should have for its objective goal the study of what kind of environment and training offers any given endowment its best chance of development. "Such inquiry takes us into the field of biological and psychobiological organization, evaluating in individual behavior the rôle of nervous system organization, glandular structure, the specific functioning of vascular, respiratory, gastrointestinal and reproductive system. These systems of the body do not function in majestic isolation, but each works as part of an exquisitely integrated organism, every cell of which has a close and subtle relationship with every other cell. The nature of this relationship is as yet very imperfectly understood, but generally speaking we think of it as having to do with nervous system mechanisms. Medical science in its efforts to study behavior of organs in their relation to the total behavior of individuals possessing these organs has gathered a considerable amount of information about what kinds of biological development stand strains of life poorly and what kind stand strains better. Inferior biological development manifests itself in a variety of ways, such as prolonged bed-wetting, night terrors, nail-biting; in such psychobiological activities as asthma, hay fever, eczema, ties, mucous colitis, cyclic vomiting, easy fatigue, etc. Medical science knows little about these conditions, treating them symptomatically, guided here and there by protein sensitization, but it is coming to realize more and more that such symptoms indicate a nervous system organization which must be carefully steered through the traffic jams of life. Prolonged physical exertion, strains of too great responsibility, environmental function which the average person meets with 'bracing up,' cause these neuropathic individuals to bend and often break."†

*November, 1932, p. 558.

†*Behavior Aspects of Child Conduct*, Esther Loring Richards, Macmillan and Company, 1932.

The physician has always realized that this array of symptomatology is found in so-called nervous children. But aside from a few general prescriptions such as taking the child out of school, or advising parents to avoid crossing him, he has paid little attention to the careful planning and supervision of the daily life of home and school which must constitute at least 50 per cent of the therapy directed toward the asthma, the eczema, and the cyclic vomiting. For example, it is just as important to work on the sensitization of the asthmatic child to the stimulations of his environment as it is to work on the problem of his sensitization to proteins; in fact the latter therapy has the worst possible chance of helpfulness without the assistance of the former. Let me illustrate. John was almost 11 when brought, a year ago, for terrific temper outbursts at home whenever crossed. During one of these he took a mouthful of iodine, swallowed a little of it, and then rushed to his mother exclaiming, "I've done it now!" The child has an interesting story of development. He is the youngest of four children. From almost infancy he showed outbursts of temper in which he would scream and strike and throw any object near his hand. He was a puny infant with whom it was hard to find food that agreed. With any emotional upset he usually vomited. His appetite was whimsical. He was particularly subject to respiratory infections, and in spite of an early tonsil and adenoid operation he was always catching cold. Between his poor food habits and his vomiting, and his being an easy prey to infections, his parents and nurses and even the family doctor, had got into the habit of getting along with him the easiest way possible. Coincident with entering a private school at six he developed asthma, and was found to be sensitive to horsehair, doghair, and feathers. In spite of regular treatment for his sensitivity and the avoidance of strenuous play and night air and draughts, the child's asthmatic paroxysms became more frequent. For over four years he had never slept in a room with the window open. He insisted on having a light burning in his room and powders by his bedside. At least once every night he had an attack, and his mother went in to him. He wet the bed until eight years of age. Intellectually the child was precocious, standing at the head of his class in school. Outside of his home he was pleasant and agreeable. He was excused from all athletic activities at school on a doctor's order. At nine years of age his father died and the family finances were so reduced as to necessitate selling the home and moving into a small apartment. The chief effect of this on John was to increase his attitude of dependence on and almost tyranny over his mother, who was obliged to work and therefore spent less time with him. He would fly into a jealous rage if she even spoke kindly to the other children. Following the iodine episode, John was removed from home and placed in an environment where he began for

the first time to control his emotions. He was not allowed to see his mother for a period of several months. He was put to sleep in a room, the windows of which were open. He had no light or powders. From this environment he was removed to the dormitory of his school, where he has lived ever since under exactly the same hygienic conditions as the other boys. During the past spring he took up horseback riding and picked ticks off the school dog. John has never had an asthmatic seizure since he began to control his emotions, and his asthmatic treatments were discontinued. This case is illustrative of the fact that it is a great mistake to allow a child with neuropathic constitution to be excused from habit training. The self-motivated stimulation of uncontrolled emotions is just as serious a factor in touching off biological instabilities as foreign proteins. One has only to look at the havoc which unrestrained impulses and instincts play in the physiologic functioning of the psychoneuroses to realize how dangerous is the procedure of letting such habit patterns get started in the biologically unstable child. It is by no means as easy to break up unwholesome emotional habits in all neuropathic children as in the case of John. When the neuropathic constitution is associated with precocious intellectual development and a personality alive with stubbornness and aggressive egoism the task is Herculean. I have in mind a certain little Polly, aged six, with the intelligence of a child of nine, who began life as a ruminator, showed eczema at two, and at five developed asthma. She became so undernourished that she developed acidosis in spite of the efforts of her parents and two nurses. She has been out of the home now for over a year in a boarding home under the care of a skillful pediatrician, who has been struggling with the problem of either keeping the child out of school to avoid infections to which she is very prone or meeting the neuropathic traits as they arise with every illness large or small which the child contracts. The asthma stopped for a period of nine months, and reappeared again during convalescence from an appendicitis and appendectomy. Following the necessity of dealing with a temper tantrum, the child will wet the bed every night for three or four nights, or start vomiting. She is fortunately in an environment and under medical management which does not allow her to utilize these symptoms wittingly or unwittingly as methods of dealing with her impulses and emotions. How many years it will take of this habit training to teach Polly the art of self-management remains to be seen. But had she remained in her former environment, a life of nervous invalidism was inevitable.

The neuropathically handicapped child is always a grave liability with regard to his ability to meet adult responsibilities thrust upon him without disaster. He is in a particularly trying position because psychology so often finds his intellectual ability excellent, and medi-

cal science finds his physiology functioning with satisfaction. We cannot explain his extreme suggestibility, his attacks of palpitation and dyspnea and fear of dying. Arguing is useless. Shaming is cruel. Coddling is fatal. Our management of each case is an individual matter depending upon a careful study of the child and environmental forces involved to determine a medium course between removal of strains and desensitization to them. All around us in these troublous times we see such children grown to maturity bending and breaking beneath loads that they are wholly inadequate to bear, carrying about accumulations of medical diagnostic suspicions, and fears contracted in childhood, and the scars and bruises of devastating experiences and vocational misadventures that it is too late to do much about now. I have in mind a husky man of 38 who has been to twenty-five physicians in the last two years for reassurance as to heart trouble. Everyone has found him sound as a bullet. It is not convincing his reason that is our job, but trying to make over years of poor mental hygiene habits and poor vocational adjustment. He is afraid to be alone, to drive a car, to lie down at night, to eat a square meal, to go up in a high building, to sit in a closed space such as moving picture house or subway. How did he get that way? He was a timid, suggestible boy, afraid of the dark, nauseated in the presence of accidents, easily fatigued by school programs that required public recitations. At ten he was taken down into a mine with a family party. He shrunk from it, but his father shamed him into going. For months afterward he walked and talked in his sleep. At fifteen he left high school because he could not face the humiliation of having been laughed at when he forgot a speech in morning assembly. Following a year at business college he drifted into office work. For the past ten years he has been secretary to a railroad manager who has found him a very efficient office buffer. He works daily from 9 to 5, and half a day on Sundays. He gets only a week's vacation every year. Doctors have advised him to relax through diversions of ball games, bridge, theatre, etc., but he is so exhausted by dinner time that he does not feel like stirring from the house. Under the strain of two pay cuts, and a decreased office force he has got into a state for which psychiatric hospitalization is necessary. He has had two outbreaks of eczema, and cannot eat a soft boiled egg without gastric discomfort. It is too late to teach this man to relax, or to tell him that he is vocationally unsuited to a railroad office. One can only tide him over successive breaks that he will undoubtedly have for the remainder of his life. Had one got him in childhood, the chance of teaching him how to live with his limitations would have been greater.

But it is not in matters of emotional self-management alone that the neuropathic constitution child finds himself strained. He is an in-

dividual who reacts to ordinary stimulations of environment that would not affect the average child. One frequently finds the tendency to biological instabilities in a serious-minded, over-conscientious, apprehensive, anxious-to-please personality. He is the sort of child whom teachers and parents overload with activities because he not only shows no resistance, but seems eager to take on responsibilities that are expected of him. At school he is encouraged to skip a grade, or to take extra subjects, and when he gets to the junior high school and high school periods he is encouraged to lead in all sorts of extra curriculum programs. Schools, and particularly progressive schools, are emphasizing qualities of leadership in students. It is a good point to stress, provided that teachers know something about the constitutional endowments of those whom they wish to feature. The child with tendencies to biological instabilities should never be urged to carry a load which makes him uncomfortable, no matter how intellectually precocious he is. Speaking in public, taking leading rôles in dramatics, competitive struggling for athletic and academic prizes are activities that are very hard for his temperament. He instinctively dreads the embarrassment and conspicuousness attendant upon such things, is inwardly consumed with a terrific fear of failure, but feels that it is his duty to do what is expected of him. The result is not infrequently a sudden outcropping of ties of choreiform movements, of stuttering or nervous fears with fatigue, loss of appetite, and general restlessness. He has no idea of what lies behind it all, and the physician finding a healthy body is apt to be equally puzzled and can only recommend withdrawal from school or a change of scene. The taking of children and adolescents out of school for nervous conditions is sometimes necessary, but in most instances it is an unwise procedure. The child gets an idea of having fallen down on his job, or his attention is fixed on "nerves" so that unconsciously he begins to keep an eye on himself from that time on. Moreover, withdrawal from school means failure to go on with the class, or a period of worry and strain associated with an attempt to make up work. It is better to keep the child in school and reduce the strains that are giving him "nerves." In the homes of these children there are usually fathers and mothers holding up standards of achievement and success that are far beyond what the child can do without a sense of discomfort. Music lessons, Hebrew school, art classes, dancing lessons are apt to fill up leisure hours that should be spent in less stimulating activities. Most parents are quite willing to prune their ambitions to suit the child's necessities if the matter is presented to them in terms of health.

Another matter that should be undertaken only after due consideration is the sending of such children to camps. A well-run camp is a godsend to many a child who needs to learn how to play with others,

to learn to live an orderly and well-arranged life in a healthy outdoor environment that he never gets a chance to experience in our crowded apartment house existence. On the other hand, physicians and schools and parents are using camps more or less indiscriminately to plan for the summer vacation of boys and girls whom they do not know what else to do with. The child with an unstable nervous and biological constitution is apt to do very poorly in summer camp unless the group is very small and those in charge of it understand such a child's needs. Camp life in large groups offers the same overstimulation that school life does with its competitive struggles, its continuous inducements to energy output, and the excitement of always being in a crowd.

In short, the health problem with a child biologically and nervously handicapped from a constitutional standpoint is the same as that of the child intellectually handicapped, in that we have to teach him early in life how to get along with his specific limitations without being self-conscious about them. It is an educational process that belongs in the field of medicine and public health. It involves parent education and teacher education as well as child education. It is a kind of educational process that cannot be achieved by physicians unless they are willing to take time and have patience to learn to inquire carefully into the lives which their small patients live at home and at school. It is the sort of education which the family doctor years ago was wont to give the community to which he ministered. He knew our constitution—what we could stand and what we would stand—and he tempered the winds of his judgment and prescriptions to meet the necessities of our individual needs.

THE HEMOLYTIC, HEMOPHILIC BACILLUS ("BACILLUS X") IN SUBACUTE BACTERIAL ENDOCARDITIS

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IN 1923 Miller and Branch[1] reported a patient with subacute bacterial endocarditis due to a hemolytic hemophilic bacillus. The patient was a twelve-year-old girl who, five months before admission, had an unexplained fever lasting two weeks and which was said to have left her with a "bad heart." Three and one-half months later she had a second attack of fever ending fatally on the fiftieth day, the last nine days of which were spent in the hospital. The patient exhibited a typical course of subacute bacterial endocarditis with fever, a systolic murmur of varying intensity, hematuria, palpable spleen, and petechiae in the skin, conjunctivae, and oral mucosa. At necropsy the patient showed, among other things, vegetative and verrucous mitral endocarditis, focal embolic myocarditis, glomerular nephritis, multiple infarcts of the spleen, kidneys, and heart, dissecting aneurysm of the abdominal aorta, and meningitis. The authors present excellent photographs of the pathologic material, among which is a photomicrograph showing the organisms in a heart valve in large numbers.

During life and from the heart valve after death they recovered a small hemolytic, hemophilic bacillus. The patient's serum agglutinated the organism in a dilution of 1:160. After a discussion of the biologic characteristics of the organism they summarized its differential bacteriologic characteristics as follows: "This organism can readily be differentiated from such other small, nonmotile, gram-negative bacilli as B. mallei, B. abortus, B. melitensis, B. pestis, and B. tularense by its inability to grow in mediums not containing blood or blood extract. It differs from B. influenzae, B. hemoglobinophilus-canis, and the Ducrey bacillus in being hemolytic. B. pertussis and the Morax-Axenfeld bacillus are not true hemophils. The hemolytic, hemophilic bacillus described by Davis[2] grew much better under anaerobic than under aerobic conditions, and failed to grow at room temperature."

Pritchett and Stillman[3] in 1919 were among the first to describe these hemolytic, hemophilic bacilli. They recovered these organisms from the throats of normal persons and grouped them under the general term "Bacillus X." Stillman and Bourn[4] later studied their cultural char-

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acteristics. Rivers[5, 7] suggested that these pleomorphic organisms might be considered hemolytic strains of influenza bacilli. As has been reported by Walker,[6] Rivers and Leuschner,[7] and Bloomfield[8] they are frequently found in the throats of normal persons and are mere saprophytes of but little pathogenic significance. Indeed, their presence in the throat is a source of considerable annoyance in doing hemolytic streptococcus carrier work because it is often very difficult or impossible to distinguish the colonies from those of the hemolytic streptococcus. Recently, Mueller and Whitman[9] have reported a cultural technic that is of value in making this distinction.

Since only one other instance has been found in the literature where this organism was the cause of a fatal illness, and this a case of subacute bacterial endocarditis, it is considered of interest to report the following case. This, in so far as we could find, is the second case to be reported. The case history is appended.

Each of four successive blood cultures (taken on the first, third, seventh, and ninth days in the hospital) and a culture from a vegetation on the mitral valve taken at necropsy yielded a pure culture of a small, pleomorphic, gram-negative, hemolytic, hemophilic bacillus. The serum of the patient failed to agglutinate the organism, but this may have been because of the relatively short duration of the illness.

Smears showed the organism to be quite pleomorphic. There were medium-sized bacilli with rounded ends and large swollen coccus forms. Considerable variation in the degree of staining was exhibited. Some of the organisms stained well, others faintly, giving a granular appearance. Stained with methylene blue, polar bodies were quite striking. The bacilli were often in pairs. Chains were occasionally noticed. After prolonged cultivation the coccus forms became much fewer in proportion to the bacillary forms. Capsules were not demonstrated by capsule stains, although some of the gram-stained specimens were suggestive. It did not closely resemble B. influenzae morphologically, in that the majority of the bacilli were larger, and there were fewer intermediate irregular forms. Repeated fishings of colonies were made to be sure two or more organisms were not present. In every instance each colony yielded the several morphologic types.

The most outstanding characteristic was the definite true hemolysis in all blood cultures. In the early isolations colonies would not appear on blood agar for three to four days, but later they grew out well in twenty-four hours. Colonies varied considerably in size. The larger colonies were grayish with a slightly yellowish tinge and were slightly mucoid in character. The zone of hemolysis about each colony was like that about colonies of hemolytic streptococci.

Virulence tests in rabbits, guinea pigs and mice were negative. All animals survived large doses. A rabbit was immunized with the organ-

ism by repeated intravenous injections and an antiserum obtained with an agglutinating titer of 1:1280. This serum failed to agglutinate B. typhosus, B. paratyphosus A and B, Flexner, Shiga, and Sonné dysentery bacilli, B. abortus, and one strain of Friedlander bacilli.

The organism was nonmotile and grew better at 37.5° C. than at 20° C. Growth at room temperature was exceedingly slow. For the first few generations at 37.5° C. it grew slowly and with difficulty. Adaptation to cultivation was relatively easy, however, and the later generations grew readily. The first few generations grew best in broth with added blood. It was only after continued cultivation that it grew readily on solid media. The best growth was obtained on solid media when the surface of the medium was first moistened with broth. After prolonged cultivation it became possible occasionally to grow the organism in broth without the addition of blood. It did not grow on other simple routine laboratory media, such as plain agar, gelatine, etc.

Fermentation tests were done using a base enriched with a small amount of hemolyzed blood. The organism fermented dextrose, maltose, and dextrine. There was no fermentation of mannite, lactose, xylose, rhamnose, saccharose, or dulcite.

As a result of these studies it was felt that this organism belonged in the group "Bacillus X" described by Pritchett and Stillman. Just how these organisms should be classified biologically, i.e., whether or not they should be considered hemolytic strains of influenza bacilli, is an academic question which we cannot discuss in this communication.

Case History.—The patient, A. P., an eleven-year-old white boy was admitted to the hospital because of a flaccid paralysis of one week's duration. Three years before the onset of the present illness he had chorea, which left him with a "damaged heart." Two weeks before admission he complained of pain in the left ankle. This was followed by pain in other joints and the development of a stiff neck. While under observation he vomited, became drowsy, in a few days became unable to speak, and a flaccid paralysis of the right side developed. He was then admitted to another hospital where he was found to have a hemiplegia on the right, a high sustained fever, and an enlarged heart with a blowing systolic murmur. The eyegrounds became progressively edematous with accompanying engorgement of the retinal vessels. This was more extensive on the right than on the left. Lumbar puncture yielded a spinal fluid under increased pressure with 250 polymorphonuclear cells per cubic millimeter and normal reduction of sugar. Organisms were not demonstrated by smear or culture. The patient was thought to have a generalized infection with embolic phenomena and was referred to this hospital* for study.

The physical examination on admission revealed an emaciated, hyperesthetic boy with a moderate degree of dehydration. The right side, including the face, was completely paralyzed. The extraocular movements were normal except for a rapid nystagmus to the left when the eyes moved in that direction. Both discs showed a slight degree of choking with engorgement of the veins. Examination of the heart showed marked enlargement to the left with an apical thrill. There was

*We are indebted to Dr. F. C. McDonald for referring this patient to this clinic.

a blowing systolic murmur over the precordium, heard best at the apex and not transmitted to the axilla. The pulmonary second sound was greater than the aortic second. The liver was tender and the edge was felt three fingers below the right costal margin. The spleen was thought to have been felt. The deep tendon jerks were obtained on the right leg but not on the arm. The Babinski sign was equivocal on the right and negative on the left.

Laboratory Data.—The urine contained a slight trace of albumin and 2 or 3 white cells per high power field. Blood examination revealed: hemoglobin 60 per cent T.; red blood cells, 2,500,000 per cubic millimeter; white blood cells, 30,000 per cubic millimeter; polymorphonuclear leucocytes, 87 per cent; lymphocytes, 2 per cent; mononuclears, 9 per cent. Numerous vacuolated phagocytes were seen. The white blood cells continued elevated throughout his course. The blood pressure was 122/70 mm. Roentgen-ray examination showed a marked enlargement of the cardiac shadow. The lungs, nasal sinuses and mastoids were clear. Lumbar

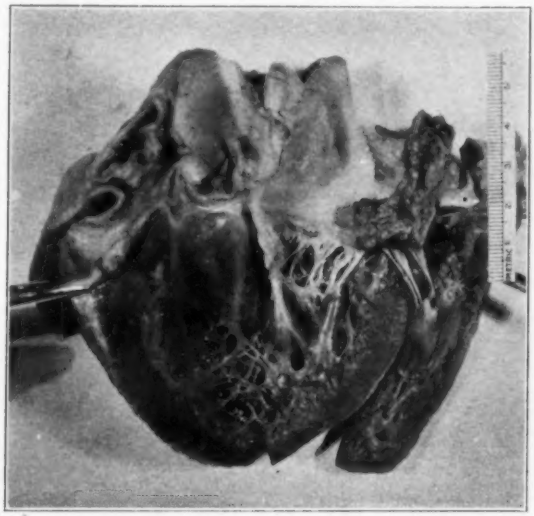

Fig. 1.—The heart with the mitral valve exposed to show the attached vegetations.

puncture on the second day yielded a clear spinal fluid with 11 mononuclear cells per cubic millimeter, negative globulin test, normal sugar, and no organisms by smear or culture. Four consecutive blood cultures were positive for "Bacillus X."

A tentative diagnosis was made of chronic rheumatic carditis and subacute bacterial endocarditis with a cerebral embolus with possibly resulting cerebral abscess.

Course.—During the first two days the blood pressure remained elevated and the optic discs continued to be choked. A hemorrhage appeared on the lateral upper margin of the right disc. Because the cerebral manifestations were thought to be caused by increased intracranial pressure due to an expanding intracranial lesion, the left parietal bone was trephined two days after admission. Direct encephalography showed both ventricles pushed over to the right. The right lateral ventricle seemed to be compressed. On the fifth day a middiastolic murmur was heard for the first time. On the sixth day the optic discs showed more choking. On the eighth day a left subtemporal decompression was done for the relief of pressure. On the ninth day petechiae were noted medial to the left scapula and

Fig. 2.—Photomicrograph of one of the small vegetations attached to the auricular surface of the mitral valve, showing the peripheral arrangement of the bacterial clumps (Giemsa, ×68).

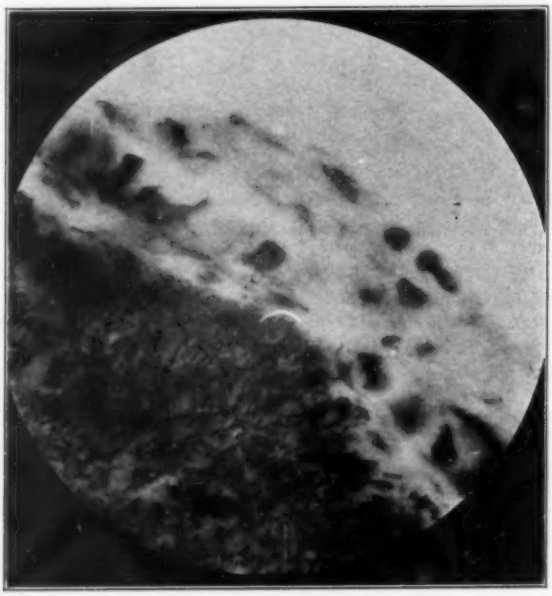

Fig. 3.—Photomicrograph showing masses of bacilli at the periphery of a vegetation. Note the thin layer of organizing fibrin covering the bacterial clumps (Giemsa ×1900).

on the buccal mucous membrane. There were râles, dullness, and tubular breathing at the right apex. Throughout his course the patient ran an irregular temperature between 103° F. and 105° F. Death occurred on the eleventh day after admission and twenty-six days after the onset of the illness.

Pathology.—Necropsy was limited to an examination of the heart. The anatomic diagnoses were: bacterial endocarditis of the mitral and aortic valves; chronic and focal myocarditis; petechial hemorrhages in the epicardium and myocardium.

The right side of the heart presented nothing unusual. Firmly attached to the anterior leaflet of the mitral valve was a large fungoid, friable, pink, fibrinous vegetation which was roughly cylindrical in shape. It measured approximately 4 cm. in length and 1.5 cm. in diameter. From its line of attachment along the margin of the valve, it extended into the cavity of the left auricle. Along the line of closure of the posterior cusp and the remaining portion of the anterior cusp, there was a series of small friable vegetations. Similar vegetations were found extending down the chordae tendineae. Along the free margins of the aortic cusps there were pinkish-gray, miliary vegetations similar to those found on the mitral valve.

Sections through various portions of the myocardium showed both acute and chronic inflammatory and degenerative changes. In certain areas there were focal accumulations of inflammatory cells between the muscle bundles which were very suggestive of the so-called Bracht-Wächter[10] bodies. Sections of the mitral valve showed it to be thickened, scarred, and having an increased vascularity. Along the margin there were discrete vegetations standing out from the surface. These contained masses of bacteria which covered most of their free surfaces. The organisms were gram-negative bacilli and showed a very slight degree of variation in size and length.

COMMENT AND SUMMARY

This, in so far as we can find, is the second case of bacterial endocarditis due to the saprophytic, avirulent organism known as "Bacillus X." The biologic characteristics of the organism isolated from this patient agree very closely with those described by Miller and Branch.[1] Whether or not the illness in this patient should be classified as acute bacterial endocarditis rather than subacute bacterial endocarditis because of its relatively short duration is questionable. Except for the duration and the bacteriology (subacute bacterial endocarditis being almost uniformly due to *Streptococcus viridans)* the symptoms, physical findings, clinical course, and some, at least, of the necropsy findings are consistent with the syndrome, subacute bacterial endocarditis.

NOTE: Since this paper was written the following additional case was seen in this clinic:

J. C., a ten-year-old Italian girl, was seen in this hospital for the first time in 1929 because of chorea following painful joints. At this time a systolic apical heart murmur was first detected. Three and a half years later (1932), after six days of malaise, fever, frontal headache and pains in the popliteal spaces and wrists, the patient was admitted to the medical wards.

The physical examination revealed a rapid heart rate, and a blowing systolic heart murmur. The liver edge was 2 cm. below the costal margin. Blood cultures yielded B. hemophilus hemolyticus on two occasions. All other cultures, repeated many times, were negative. The patient's serum agglutinated this organism in dilutions up to 1:20. The course was typical of bacterial endocarditis with the

following features: A swinging temperature up to 104° F. to 105° F., a polymorphonuclear leucocytosis, the development of an apical diastolic heart murmur, rare red blood corpuscles in the urine, numerous embolic phenomena including petechiae, Osler nodes, emboli to the spleen, brain and left leg. The patient died after thirty-eight days on the ward; forty-four days after the onset of the terminal illness. Autopsy permission was not obtained.

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INFECTIOUS (SUBACUTE BACTERIAL) ENDOCARDITIS WITH CEREBRAL THROMBOSIS

Report of a Case

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THIS disease "consists of the invasion of the heart—chiefly of the valves—by the Streptococcus viridans, rarely by the gonococcus or influenza bacillus; it almost always results fatally after a long illness." It occurs most commonly between the ages of fifteen and thirty years.[1] The literature on the disease in infancy was reviewed by Sansby and Larson in 1930.[2] Rost and Fischer[3] in 1928 reviewed the reports of this disease in children. They collected 64 cases under fourteen years of age, added 12 of their own, and 4 of J. D. Nourse. Lawson and Palmer[4] reviewed the literature and added 3 cases of six, eight, and ten years of age, respectively. Leech[5] added 15 cases of Streptococcus viridans endocarditis under fifteen years of age. He found paresis in 25 per cent. Cerebral complications are not unusual. Rheumatic fever is the chief predisposing factor.

The following case is reported because of the character of the onset, the long period of observation, and the necropsy findings.

History.—S. K., a girl seven years of age, was admitted December 1, 1931, because of inability to use the right upper and lower extremities. She had been in perfect health until three and one-half months previously when she began to have nosebleed about once a week. She was examined with her chest bare by a school physician in September. He pronounced her in good health and free of heart disease. Two weeks later she had a severe hemorrhage from the nose which lasted four hours. Following this, the child became pale and was not as well as previously. She tired easily and was anxious to go to bed early. One month after the onset, there occurred another severe nasal hemorrhage which lasted three hours. No hemorrhages were noted elsewhere in the body. The child had no further bleeding until a week before admission when there was another severe nasal hemorrhage. Five days before admission, the child complained of pain in the lower left chest. Two days later she was pale and listless and went to bed at 8:00 P.M. When she awakened the next morning at 8:00 o'clock, she was unable to move her right arm and leg.

Physical Examination.—Examination on admission revealed an acutely ill, irritable, well-developed, fairly well-nourished, pale, drowsy girl, who seemed unable to talk. The essential findings were: Rectal temperature 101° F., respiration 28, pulse 130. The right cheek was flushed, and there was a central facial palsy of this side. The fundi were negative. The heart was slightly enlarged, with a diffuse, forceful apex beat at the nipple line in the fifth, left, intercostal space. There

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was a loud, blowing, systolic murmur heard best over the apex and transmitted to the left axilla. Pulse rate was 130 and blood pressure 110/80. The liver edge was tender and just palpable below the costal margin. The spleen edge was felt two centimeters below the costal margin. The superficial abdominal reflexes were absent on the right. There was a flaccid paralysis of both right extremities, the deep reflexes were absent, and there was a positive Babinski reflex and ankle clonus. No petechiae were found. A bruit was heard over the entire head, more marked over the right side (Dr. J. L. Garvey). The blood findings were as follows: Hb. 35 per cent, R.B.C. 2,390,000, marked anisocytosis and poikilocytosis, W.B.C. 6,200, neutrophiles 68 per cent, small lymphocytes 20.5 per cent, large lymphocytes

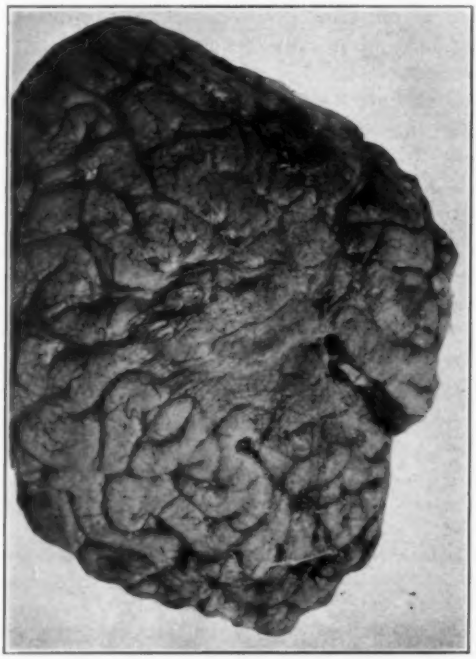

Fig. 1.—Brain, showing thickened pia over right Rolandic area.

9.5 per cent, mononuclears 0.5 per cent, platelets 226,000, coagulation time (Bogg's) 3 minutes, bleeding time ½ minute, Lee coagulation time 9 minutes, calcium time 7 minutes, clot retractility 2½ minutes, prothrombin time

2	3	4	5	6	7	8	drops $CaCl_2$
10	10	10	12	14	14	14	min.

Blood culture showed no growth in seventy-two hours. Wassermann and micro-precipitation tests were negative for syphilis. Urinalysis was negative. The stools were normal.

Dec. 5.—The blood count was Hb. 42 per cent, R.B.C. 2,610,000, W.B.C. 7,200 with neutrophiles 60.5 per cent, small lymphocytes 31 per cent, large lymphocytes 6.5 per cent, large mononuclears 1.5 per cent, eosinophiles 0.5 per cent. The Schilling count showed 48.5 segmented forms, 10.5 staff forms, and 1.5 young

forms. Fifteen cubic centimeters of clear spinal fluid under normal pressure was obtained by lumbar puncture. It showed one cell, no globulin, 63 mg. sugar, a negative Lange gold sol, and no organisms on culture. Wassermann and micro-precipitation tests were negative. On the same day a transfusion of seventy-five cubic centimeters of whole blood was given.

Dec. 7.—Roentgenogram of the chest (6 ft. film) chest width 18.1 cm., heart width at apex 10.5 cm.,—definite enlargement.

Dec. 8.—Hess capillary resistance test negative. Sedimentation rate greatly increased (Westergren ½ hr. 15 min., 1 hr. 25 min., 2 hr. 50 min., 24 hr. 180 min.). Blood culture was negative after forty-eight hours.

Dec. 9.—There was a small bleeding point on the left side of the pharynx behind the posterior pillar. A large clot of dried blood was removed from the left

Fig. 2.—Section through area shown in Fig. 1, showing softening.

nostril and there was bleeding from a point on the anterior of the left side of the septum (Dr. E. R. Ryan). The patient showed slight improvement and answered simple questions.

Dec. 15.—Condition about the same. Daily fever to 101.2° F. Right hemiplegia and facial paralysis the same. Cerebral bruit heard only over left eye. Hb. 48 per cent, R.B.C. 3,800,000, W.B.C. 10,000, neutrophiles 64 per cent, small lymphocytes 27 per cent, large lymphocytes 9 per cent.

Dec. 17.—Urine acid; specific gravity 1.025; albumin 1; pus 18 cells in high power field.

Dec. 18.—Blood culture forty-eight hours, Streptococcus viridans. Cultures of blood taken Dec. 7 and 9 show no growth after nine and ten days, respectively.

Dec. 21.—Condition the same. Urine: pus, 25 cells in high power field.

Dec. 24.—Blood culture—Streptococcus viridans.

Dec. 26.—Blood culture—Streptococcus viridans.

Dec. 28.—Hb. 55 per cent, R.B.C. 3,380,000, W.B.C. 7,800, neutrophiles 60.5 per cent, small lymphocytes 37 per cent, large lymphocytes 2.5 per cent.

Jan. 1, 1932.—Small petechia on conjunctiva of each lower lid.

Jan. 2.—Able to move right upper extremity for first time since admission. Pyuria continues.

Jan. 5.—Heart condition the same. Slight improvement in use of right arm and in speech. Irregular evening temperature increased to 103° F. (rectal). Electrocardiogram normal.

Fig. 3.—Heart, showing vegetations on mitral and tricuspid valves and on the wall of the left auricle.

Jan. 14.—Blood culture negative forty-eight hours.

Jan. 16.—Hb. 50 per cent, R.B.C. 3,150,000, W.B.C. 4,800, neutrophiles 50 per cent, small lymphocytes 32 per cent, large lymphocytes 10 per cent, myelocytes 4 per cent. Rectal temperature at 8:00 P.M. 105.2° F. General condition not as good. Developing contracture deformities of right extremities.

Feb. 8.—Hb. 45 per cent, R.B.C. 3,680,000, W.B.C. 8,000, neutrophiles 60 per cent, small lymphocytes 26 per cent, large lymphocytes 13 per cent, transitionals 1 per cent. More listless, tender to touch in both lower extremities. Blood pressure 112/80. Afternoon elevation temperature continues. General condition not as good.

March 1.—Urine; acid, specific gravity 1.010, Albumin iii, R.B.C. 25 cells, pus cells 10 in high power field. General condition poor; stuporous, refuses food.

March 5.—Albuminuria and hematuria continue. Marked café au lait pallor, face puffy, marked hypersensitivity of extremities and abdomen; finger tips cyanotic.

March 7.—Hb. 50 per cent, R.B.C. 3,480,000, W.B.C. 16,400, neutrophiles 78 per cent, small lymphocytes 11 per cent, large lymphocytes 10 per cent, monocytes 1 per cent, anisocytosis, poikilocytosis, and achromasia.

March 8.—Blood culture—Streptococcus viridans. Albuminuria, hematuria, and pyuria. Finger tips clubbed and cyanotic.

March 13.—Patient died.

Necropsy.—The following were the important findings: There was a contracture deformity of the right arm and leg. There was an area of beginning atrophy and softening over the left Rolandic area of the brain, measuring 6 by 5 cm. The pia was thickened and fibrous over this area. The cortex was softened for 1 cm. below the pia. There was a thrombus in the left posterior cerebral and posterior communicating arteries near their origin. (See Figs. 1 and 2.) Both lungs showed congestion. There was 125 c.c. free, thin fluid in each pleural cavity. The heart weighed 225 grams and measured 11.5 cm. in its greatest transverse diameter. There was an infarct 0.6 cm. in diameter in the wall of the left ventricle. Large vegetations were found on both cusps of the mitral valve and on the wall of the left auricle. There was a small vegetation on the wall of the tricuspid valve. (See Fig. 3.) There was no free fluid in the peritoneum. The liver weighed 1015 grams and was pale in appearance. On microscopic section it showed some fatty degeneration, chronic passive congestion, and early cirrhosis. The spleen weighed 220 grams, was firm, and had four infarcts. There was a small hemorrhage in the right suprarenal. The right kidney weighed 120 grams, left 135 grams, and both were covered with petechiae on the surface. There was a large petechia on the surface of the right kidney and several small infarcts in both. On microscopic section there was degeneration of many of the glomeruli.

This rather typical case of subacute bacterial (infectious) endocarditis is reported because of the cerebral complication, a long period of observation, confirmation of the diagnosis by blood culture, and by subsequent necropsy.

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A CRITICAL CLINICAL STUDY OF CONCENTRATED AND DRIED INFANT FOODS

II. Modified Dried Milk

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THIS is the second of a series of articles in which we are examining clinically the merits of different infant foods. Our first article treated the use of condensed milk.[1]

REASONS FOR INVESTIGATION

Various powdered milks have been in use for the past forty years. The literature is fairly rich in attesting to the safety, the uniform results, and the merits of these. The local Government Board of Great Britain[2] in 1918 made an extensive survey of the use of dried milk in infant feeding and concluded that when breast milk is not available dried milk is a very valuable food for infant feeding. Aviragnet and Dorlencourt[3] in 1922 published the results of approximately fifteen years of experience and came to the conclusion that their findings confirmed the view that a powdered milk of good quality constitutes an extremely favorable food for the artificial feeding of infants. They reported that it may be used as a complete and sole food for infants in this respect, it being neither superior nor inferior to a milk of good quality. These general views are upheld in the clinical works of Millard,[4] Naish,[5] Hill,[6] Still,[7] Ashton, et al.,[8] Abt and Feingold,[9] Gerstenberger,[10] Ribadeau[11] as well as others who have shown the advantage and successful use of dried milks in functional vomiting, indigestion of cow's milk, in diarrheas, athrepsia and anaphylaxis to cow's milk.

Emerson[12, 13] has done some interesting work in the successful feeding of dried human milk to premature infants and those with severe intestinal intoxication. McCord[14] has reviewed the literature of the subject of dried foods in a broad manner and showed the uniform good results with its use.

By virtue of the fact that most of the powdered milks have been given clinical investigations by the aforementioned authors, we decided to make a clinical study of a popularly used American modified dried milk.

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METHOD OF MANUFACTURE

The drying of milk began at about the same time as condensing, but it is only within the last two decades that powdered milk has been manufactured on a successful scale.[15] Grimwade secured a British patent in 1855 for a process in which carbonate of soda was first added to the whole milk, followed by evaporation in open pans to a paste, then the addition of cane sugar, with final pulverization between rollers. In 1872 a method for drying milk by spraying it into a chamber of hot air was patented, but the first really successful method was invented by a German, Robert Stauf, who obtained a patent in 1901. Since then various inventors notably the Merrell Brothers and John A. Just have invented and patented processes for desiccating milk on heated rollers and by spray processes.

In the spray process, tested milk is first pasteurized and then condensed in vacuum in order to reduce the water content. It is then forced under high pressure through fine nozzles into a metal chamber heated to a temperature of about 270° F. The moisture evaporates and rises and the remaining powder collects at the bottom of the chamber, from which it is removed at once and placed in the containers.

In the roller process whole milk is condensed and then poured upon revolving steel drums heated to a high temperature. The milk dries immediately and is automatically scraped from the drum into receptacles and then placed in cans. Both of these processes are rapid, consuming only a few seconds.

That heat treated milk is more digestible by pepsin in vitro than raw milk has been shown by Hess, Koch and Sennewald[16] and substantiated clinically by Abt[17] and Dennett.[18] The heat treated milk curd is a softer broken curd and the greater digestibility may lie in the exposure of a larger surface to the digestive juices and in the denaturation of the proteins.

DIGESTIBILITY AND NATURE OF PRODUCT

The dried milk used in this study is made by the spray process. In speaking of the solubility of dried milk made by the spray process Hunziker[19] states that drying processes that expose the milk to high temperature by direct contact with steam heated surfaces, as is the case in the film-drying process with steam heated rolls, produce milk powders with relatively low solubility. The metal surface of the steam heated drums reaches in most of the film-drying processes a temperature considerably above the boiling point. The temperature used in the Just-Hatmaker process, for instance, is well above the boiling point. In practical operation a temperature of approximately 270 degrees F. is attained. Tests show that the solubility of milk powders from high temperature drum driers is relatively low.

In the preparation of the infant food used in this study whole milk is modified by the addition of cream from fresh milk and of lactose. The mixture is put through the described spray process and the forthcoming cool powder is packed in an atmosphere of nitrogen gas and hermetically sealed. In view of the interest recently awakened in soft curd milk and in the curd tension of various infant foods generally, it is well to mention the subject of the curd. It is a well-known fact that the curd of cow's milk forms in a tough mass which varies considerably from the soft flaky curd obtained from human milk. Hill has

shown that there is a range of from 15 to over 200 grams in the curd tension of the milk from different cows. In view of the fact that the curd character of milk is an individual characteristic of the cow and is fairly uniform and apparently permanent, the use of milk with a low and uniform curd tension avoids the necessity of the selection of individual milks from various cows. Raw milk and only to a somewhat lesser extent pasteurized milk or even boiled milk coagulates in relatively large and heavy curds soon after reaching the infant's stomach. The dried milk, on the other hand, forms curds of markedly smaller size with a very low curd tension, and is ideal for the assumption that the smaller the size of the curds the more surface is exposed to digestive action, and hence the more rapid and complete is the digestion.

The heating periods, together with effects produced by the passage of the concentrated milk through the homogenizer and the drying of the small particles of milk solids in the spray chamber, slightly disrupt the original colloidal system of the liquid milk. After reconstitution with water the casein portion when precipitated by the acid fluid elaborated in the infant's stomach does not form the heavy masses of curd produced by raw pasteurized or even boiled milk but instead forms small soft flocs. Consequently the protein is easily digestible.

THE CHEMICAL COMPOSITION

The chemical composition of the milk powder is shown in Table I.

TABLE I

Moisture	2.0%
Ash	3.5%
Protein (Nx6.38)	16.2%
Fat (ether extract)	25.0%
Lactose (by difference)	53.3%
Calories 5.02 per gram or 142.96 per ounce.	

For practical feeding use, one can take three teaspoonfuls to equal one tablespoonful and four tablespoonfuls to equal an ounce by weight. One tablespoonful can be used as equal approximately to 40 calories.

This food has a high fat content. Holt in his book, *Food Health and Growth*, speaking on the subject of fat requirements in the diet of infants states that although some investigators are convinced that there is no relation between the fat intake and the mineral metabolism in general, he feels certain that in the case of calcium metabolism at least, a very definite relation exists. The work on calcium metabolism done in the laboratory of the Babies Hospital in New York indicated that the best calcium absorption takes place when the fat in the diet bears a certain relation to the calcium and when the intake of both is liberal.

TABLE II

FORMULAS OF DRIED FOOD USED

Twenty-Four-Hour Quantity

	DRIED FOOD GRAMS	VOLUME C.C.	GRAMS DAILY				TOTAL WEIGHT DAILY		PERCENTAGE BY WEIGHT				APP. CALORIC VALUE PER OZ. OF MIXTURE
			FAT	PROTEIN	LACTOSE	ASH	LACTOGEN	WATER	FAT	PROTEIN	LACTOSE	ASH	
1st day	10	120	2.5	1.6	5.3	0.35	130		1.9	1.25	4.1	0.27	13
2nd day	15	180	3.75	2.4	8.0	0.53	195		1.9	1.25	4.1	0.27	13
3rd-4th day	26.25	315	6 5	4.25	14.0	0.92	341		1.9	1.25	4.1	0.27	13
5th-7th day	37.5	315	9.4	6.1	20.0	1.31	352.5		2.6	1.7	5.6	0.38	18
2nd week	60.0	525	15.0	9.7	32.0	2.10	585		2.55	1.65	5.5	0.36	18
3rd week	78.75	630	19.7	12.7	41.9	2.75	709		2.8	1.8	5.9	0.39	19
4th week	97.5	735	24.4	15.8	52.0	3.42	832.5		2.9	1.9	6.2	0.41	20
2nd month	105.0	840	26.3	17.0	56.0	3.68	945		2.8	1.8	5.9	0.39	20
3rd month	131.25	1050	32.8	21.3	70.0	4.60	1181		2 8	1.8	5.9	0.39	20
4th month	135.5	1080	33.9	21.9	72.3	4.75	1215		2.8	1.8	5.9	0.39	20
5th month	150	1050	37.5	24.3	80.8	5.26	1200		3 12	2.02	6 66	0.44	22
6th-12th month	150	1050	37.5	24.3	80.0	5 26	1200		3.12	2.02	6 66	0.44	22

That fat is an important component of the diet of the infant is shown by the composition of human milk. The normal amount of fat in breast milk averages between 3 and 4 per cent and this furnishes approximately half the total calories in the diet of the nursing infant. It seems hardly conceivable that so much fat would be supplied by nature if the only specific purpose of the fat were to furnish vitamin A.

The nursing infant usually receives during the early weeks as much as 20 grams of fat daily. This total increases by the seventh month up to an average of 40 grams daily. This represents about 4 grams per kilo of body weight.

The food used in this study has been designed to include a high proportion of milk fat, as it is modelled on breast milk as a standard and the average fat content of breast milk is 3.0 to 4.0 per cent. Fat is the only natural conveyor of the fat-soluble vitamins A and D, and the vitamin supply in the case of an infant given a cow's milk formula is in direct proportion to the amount of fat given in the food; that is, if a cow's milk containing 3.6 per cent of fat has a vitamin content of X units, a formula made up from this milk and having a fat content of 1.8 per cent fat will have a vitamin content of only x/2 units, while if so arranged as to have a fat content of 3.6 per cent it will convey X vitamin units.

Fat in the food aids the digestion of protein, the absorption of protein being low when the fat is low.

This holds true also with the absorption and retention of calcium.

Fat is economical physiologically, providing over twice as many calories per gram as do carbohydrates and protein and thus increasing the food value of a feeding without unduly increasing bulk.

The fat of the infant's food is almost completely utilized, from 90 to 99 per cent of the intake being normally metabolized. It has been found by experiment that more complete utilization occurs when the proportion of fat in the food is high than when it is low. There are comparatively few instances on record in which the digestion of fat was so poor that large amounts of fat were excreted in the stools unless fermentative diarrhea due to a disturbance of carbohydrate digestion had preceded.

There is a common fallacy about fat digestion which results from insufficient knowledge. This is that the fat loss in the stools on a high fat diet is excessive because the proportion of fat found in the stool is large. When correlated with the total daily amount of stool and the daily fat intake it is invariably found that the total amount of fat utilized is very large, usually over 95 per cent of the intake. It is the amount of fat which is utilized which is important, not the amount of fat passed in the stools. For instance, if an infant receives 15 grams of fat daily and excretes 1.0 gram in the stool, the child is utilizing

14 grams daily. If the infant receives 30 grams of fat daily and excretes even 3.0 grams, or 10 per cent, which rarely happens, the amount utilized is 27 grams.

Another common fallacy is that a generous proportion of fat in the food induces vomiting. Many investigators have found that this is not the case as long as the milk fat is processed and homogenized. Very few infants develop vomiting due to fat indigestion when proportions of fat similar to those found in human milk are given, and most infants can successfully digest much higher proportions of homogenized and processed milk fat. Striking evidence concerning this is found in the fact that two very successful therapeutic foods used in cases of extremely delicate digestive conditions, protein milk, used for diarrhea, and butter-flour mixture, used for marasmus and for premature infants, are both foods high in fat and the use of these is not usually followed by vomiting.

COMPARATIVE ANALYSIS OF VARIOUS FORMULAS

By disregarding the water content in an infant formula the food constituents expressed as proportions of the solids in the mixture is interesting. The addition of water to any food mixture does not change the quantitative or caloric relationship of the food components to each other. A very different result, however, is obtained when milk is diluted not with water but with an energy-yielding substance. Carbohydrates, for example, when added in increasing amounts to a fixed volume of whole cow's milk assume an increasing percentage representation in the total energy value of the mixture at the expense of every other food component—protein, fat, and carbohydrate. As Powers has said they are "squeezed by the added carbohydrate to smaller and smaller percentage of the total caloric valuation. In the

TABLE III

32 OUNCES (⅓ MILK – ⅔ WATER) 1 OUNCE OF SUGAR – TOTAL 33 OUNCES

	WET BASIS %	DRY BASIS %
Fat	1.18	16.56
Protein	1.12	15.65
Lactose	1.59	22.22
Ash	0.22	3.18
Sugar	3.03	42.39
Water	92.86	----

TABLE IV

32 OUNCES (½ WATER – ½ MILK)

	WET BASIS %	DRY BASIS %
Fat	1.83	28.75
Protein	1.74	27.25
Lactose	2.45	38.58
Ash	0.35	5.42
Water	93.63	----

TABLE V

32 OUNCES (½ MILK – ½ WATER) 1 OUNCE SUGAR – TOTAL 33 OUNCES

	WET BASIS %	DRY BASIS %
Fat	1.77	19.24
Protein	1.68	18.26
Lactose	2.38	25.87
Ash	0.34	3.69
Sugar	3.03	32.94
Water	90.80	----

TABLE VI

32 OUNCES (½ MILK – ½ WATER) 1.5 OUNCES SUGAR – TOTAL 33.5 OUNCES

	WET BASIS %	DRY BASIS %
Fat	1.74	16.48
Protein	1.65	15.72
Lactose	2.34	22.25
Ash	0.33	3.13
Sugar	4.48	42.42
Water	89.46	----

TABLE VII

32 OUNCES (⅔ MILK – ⅓ WATER)

	WET BASIS %	DRY BASIS %
Fat	2.44	28.74
Protein	2.31	27.21
Lactose	3.28	38.63
Ash	0.46	5.42
Water	91.51	----

TABLE VIII

32 OUNCES (⅔ MILK – ⅓ WATER) 1 OUNCE SUGAR – TOTAL 33 OUNCES

	WET BASIS %	DRY BASIS %
Fat	2.37	21.05
Protein	2.24	19.89
Lactose	3.17	28.15
Ash	0.45	4.00
Sugar	3.03	26.91
Water	88.74	----

TABLE IX

32 OUNCES (⅔ MILK – ⅓ WATER) 1½ OUNCE SUGAR – TOTAL 33.5 OUNCES

	WET BASIS %	DRY BASIS %
Fat	2.33	18.52
Protein	2.20	17.49
Lactose	3.13	24.88
Ash	0.44	3.50
Sugar	4.48	35.61
Water	87.42	----

TABLE X

32 OUNCES (⅔ MILK – ⅓ WATER) 2 OUNCES SUGAR – TOTAL 34 OUNCES

	WET BASIS %	DRY BASIS %
Fat	2.30	16.58
Protein	2.18	15.72
Lactose	3.08	22.21
Ash	0.43	3.10
Sugar	5.88	42.39
Water	86.13	----

TABLE XI

DRIED MODIFIED MILK FORMULAS
6 OUNCES WATER PLUS 2 TABLESPOONFULS DRIED MODIFIED MILK

	WET BASIS %	DRY BASIS %	GRAMS DAILY
Milk Fat	1.90	25.51	3.75 grams
Milk Protein	1.25	16.53	2.40 "
Milk Sugar	4.10	54.39	8.00 "
Milk Salts	0.27	3.57	0.53 "
Water	92.48	----	
	100	100	

TABLE XII

10½ OUNCES WATER PLUS 5 TABLESPOONFULS DRIED MODIFIED MILK

	WET BASIS %	DRY BASIS %	GRAMS DAILY
Milk Fat	2.60	25.51	9.40 grams
Milk Protein	1.70	16.53	6.10 "
Milk Sugar	5.60	54.39	20.00 "
Milk Salts	0.38	3.57	1.31 "
Water	89.72	----	
	100	100	

TABLE XIII

21 OUNCES WATER PLUS 10½ TABLESPOONFULS DRIED MODIFIED MILK

	WET BASIS %	DRY BASIS %	GRAMS DAILY
Milk Fat	2.80	25.51	19.7 grams
Milk Protein	1.80	16.53	12.70 "
Milk Sugar	5.90	54.39	41.9 "
Milk Salts	0.39	3.57	2.75 "
Water	89.11	----	
	100	100	

TABLE XIV

35 OUNCES WATER PLUS 20 TABLESPOONFULS DRIED MODIFIED MILK

	WET BASIS %	DRY BASIS %	GRAMS DAILY
Milk Fat	3.12	25.51	37.50 grams
Milk Protein	2.02	16.53	24.3 "
Milk Sugar	6.66	54.39	80.8 "
Milk Salts	0.44	3.57	5.26 "
Water	87.76	----	
	100	100	

dried food used in this study the dried whole milk reconstructed by the addition of carbohydrate as well as fat still retains its higher fat percentage even in considering it from this highly technical angle.

It is generally assumed by pediatricians that infants cannot tolerate as high a percentage of fat in a modified milk formula as they can in breast milk. Experience has taught us, however, that this does not hold true in the case of heat treated and homogenized milks, such as used in this study, the process of heating and drying rendering the fat more easily digestible.

VITAMINS

The present controversy on the subjects of vitamins in dried milk has centered itself on the relative antiscorbutic and antirachitic content in these products. It is generally conceded that with any milk, whether dried or not, antiscorbutic and antirachitic agents are required. The thermostability of vitamins A and B has been well established by Osborne and Mendel,[20] Steenbock, Sell and Buell,[21] Roseneau,[22] and Slanetz.[23] Drummond[24] states that the processes involved in the preparation of dried milk does not appreciably affect the A and B vitamins. Sherman, et al.,[25] in experimenting with rats when milk was replaced by dried milk, reported no serious destruction of either fat-soluble A or water-soluble B. Hooker and Johnston[26] showed that the process of drying milk does not lead to an appreciable destruction of the antineuritic vitamin.

Data from different laboratories[27, 28, 29, 30, 31] show that the antiscorbutic property of milk is not of constant and uniform potency. Various writers[32, 33, 34, 35] have shown that these wide variations in the vitamin C content of milk is due to the differences in ration fed during the period of lactation, in the summer and winter, as well as to the contamination of milk by small amounts of copper during pasteurization.

Cavanaugh, et al.,[36] and Supplee and Dow[37] as well as others[38, 39, 40, 41] disagree as regards the value of the protective ability of milk powders made by different methods. Some claim and show that the spray process depletes the vitamin C content of the milk powder while others point out the same in the roller process. Although the foregoing findings are interesting, the addition of an active antiscorbutic agent eliminates the necessity of any scientific controversy.

BACTERIOLOGY

The following tests were made for the purpose of comparing the numbers of bacteria found in milk formulas prepared with fluid milk and a popularly used maltose and dextrin carbohydrate, and milk formulas prepared with water and the dried food used in this study.

Quart bottles of certified and Grade A pasteurized milk were purchased from drivers of two of the large representative milk concerns in this city and a can of the carbohydrate and three cans of the dried food used here were purchased on the open market at three different stores.

The preparation of the formulas was carried out as it would be done in the home. The glass measuring and mixing dishes were scalded before use. Spoons, bottles, and stoppers were boiled. Tap water was boiled fifteen minutes and cooled for use in the formulas. Bottles were boiled and cooled before being filled. The formulas using fluid milk were prepared as follows:

Milk	21 fluid ounces
Water	11 fluid ounces
Carbohydrate	1¼ ounces (6 level tablespoonfuls)

Four samples were prepared from this formula using Company No. 1 certified, Company No. 2 certified, Company No. 1 Grade A pasteurized and Company No. 2 pasteurized milk.

The formulas containing the dried food used in this study were made as fololws:

Dried food	1 ounce
Water	7 ounces

Three samples, using the dried food from three cans purchased at three different stores were prepared.

Examinations to determine the number of bacteria capable of growing on standard beef extract agar, incubated two days at 37° C., were made on the four bottles of fluid milk used in preparing the formulas, and examinination was made at once on all of the seven samples. The samples were then placed in a refrigerator where the temperature was 44° to 46° F. The samples were tested again after six hours and then after twenty-four hours and forty-eight hours, being left meanwhile in the refrigerator.

The results of the examinations are given in Table XV.

The average bacterial count of the dried milk used runs between 2000 and 2500 per gram as determined by the standard plate method of the American Public Health Association. The bacteria have been found to be harmless spore-forming organisms, such as the hay and potato bacillus natural to fresh milk. None of these are pathogenic. In examining 12 samples of this dried milk bacteriologically Giblin and von Pourtales[42] found the average plate counts incubated at 37° C. to be 6000 (number of bacteria per gram) and the cultures for B. tuberculosis and Streptococcus viridans and hemolyticus to be sterile. The guinea pig inoculation for B. tuberculosis was also negative. The

TABLE XV

BACTERIAL COLONIES PER C.C. GROWING ON BEEF EXTRACT AGAR – 2 DAYS 37° C.

FORMULA	DEALER AND GRADE MILK USED	MILK USED IN FORMULA	AT ONCE	AFTER 6 HOURS TEMP. 46° F.	AFTER 24 HOURS TEMP. 44° F.	AFTER 48 HOURS TEMP. 45° F.
Milk 21 oz. Water 11 oz. Carbohydrate 1½ oz.	Dealer No. 1 Certified	3000	2100	2700	3900	4500
"	Dealer No. 2 Certified	3200	2200	3300	2700	840
"	Dealer No. 1 Grade A Pasteurized	1400	800	760	3200	12000
"	Dealer No. 2 Grade A Pasteurized	3500	2400	2300	2900	7200
Dried Food 1 oz. Water 7 oz.	Purchased Store No. 1	–	1500	1900	2500	1900
"	Purchased Store No. 2	–	2300	3800	6200	16000
"	Purchased Store No. 3	–	730	1200	1300	3300

average bacterial count per gram of 20 other dried foods they examined ranged from 2000 to 10,000.

In a study undertaken in connection with an extensive investigation on the bacteriology of prepared infant foods to determine the prevalence of hemolytic streptococci in such foods Hucker and Hucker[43] found on examination of a representative series of them no appreciable types of streptococci.

CLINICAL INVESTIGATION

One hundred and forty-four infants of which 117 are reportable were fed the dried milk as a routine food. The other 29 cases were dropped or not reported for various causes, such as lack of cooperation, not on food long enough, and failure to return to the clinic.

Method of Feeding.—The infants were fed according to their caloric requirements, after the method of Dennett.[18] The fat infants over four months of age received from 40 to 50 calories per pound of body weight; the average infants under four months of age and moderately thin infants of any age received from 50 to 55 calories per pound of body weight, and the emaciated infants received from 60 to 65 calories per pound of body weight.

Age.—The earliest age at which any infant was put on the food was 2 weeks and the latest 3 months. The average age at which it was started was 2 months. In the athreptic group the youngest infant was 1¾ months and the oldest 6 months, the average age being 2½ months.

Time of Observation.—The average length of time each infant was on the food was 23 weeks. The average age at which the food was stopped

was 7¼ months. In one case it was continued until the child was a year old. Although it is the customary practice to discontinue the use of this food at the usual time of weaning the child from the breast at about 10 months of age, there is no reason for not continuing it until the child is a year old or more, as was the case in some of our reportable cases. Indeed, we have fed children of school and preschool age on this food, giving it to them because of economic conditions in the home. The results were invariably good. The average age at which food was stopped in this group was 6.6 months and no case was observed for less than 6 weeks.

Types of Cases.—Of the 117 reportable cases 100 were infants who were of normal weight for their age and 17 were athreptic infants and are reported separately as such. Of the 100 normal infants the dried milk was used as a complemental feeding in 11 cases for an average of 3.4 weeks before going on to a schedule of total artificial feeding.

Weight.—The average gain in ounces per week of the normal infants was 5.45 and of the athreptic infants 5.4 ounces. The average gain per week of the complemental cases was 6 ounces.

These figures are an average of the total number of cases regardless of age. They compare favorably with the average series of cases in the literature. Stimson[44] observed 53 infants on a diet of milk, water, and added carbohydrate and showed that they gained an average of 1.799 ounces a week; later he observed 57 infants of the athreptic type on a similar diet. These gained an average of 1.365 ounces a week. Marriott and Schoenthal[45] reported 67 infants fed unsweetened evaporated milk in out-patient and office practice who gained 6.2 ounces weekly while 75 hospital cases averaged 4.55 ounces. Jarvis[46] in observing infants fed on milk, water, and lactose found that 64 under six months of age gained an average of 5.75 ounces per week and 36 infants over six months gained an average of 4.5 ounces per week.

In a group of 60 breast-fed infants used as a control the average gain was 5.55 ounces weekly.

Gastrointestinal Upsets.—Among the 100 normal infants there were only 3 gastrointestinal upsets. They consisted of eight to ten yellow, semisolid stools a day and the condition cleared in from two to four days by simple reduction in the food. In the series of 17 athreptic infants there was one gastrointestinal upset consisting of 7 to 8 yellow, semisolid stools for two days; this case was also cleared by the simple reduction of food. One would expect this number of gastrointestinal upsets in a similar series of cases being fed any type of food.

Infections.—Among the normal babies there were 6 cases of upper respiratory infection and 4 cases of otitis media that resolved without complications. In the athreptic group there were 2 cases of upper

respiratory infection, both with complicating otitis media. Compared with control cases this incidence of infection shows no increase over that of infants fed by other foods.[1]

TABLE XVI

INCIDENCE OF ACUTE INFECTIONS

	DRIED FOOD IN THIS STUDY	MILK, WATER AND CARBOHYDRATE	CONDENSED MILK
Otitis media	4 (4%)	4 (8%)	3 (6%)
Upper respiratory infection	6 (6%)	2 (4%)	2 (4%)
Mastoiditis (Child had chronic purulent otitis media when put on feeding)	0	0	1 (2%)
Number of cases reported on	100	50	50

Organic Abnormalities.—In this group of athreptic infants there was one microcephalic child, one case of harelip and cleft palate, and one child who developed acute poliomyelitis after it had been on the feeding three months.

Deaths.—There were two deaths in the athreptic group: one, an infant having an upper respiratory infection followed by pneumonia and the other, an infant with harelip and cleft palate that did well until 5 months of age but developed acidosis after operation and died. A general summary of the results follows:

NORMAL CASES—100

Average birth weight—7 pounds, 10 ounces
Average age when food was started—2 months
Earliest age started—2 weeks
Oldest age started—3 months
Average age food stopped—7¼ months
Oldest age food stopped—1 year
Average time on food—23 weeks
Shortest period on food—6 weeks
Longest period on food—42 weeks
Average weight when food was started—10 pounds
Average weight when food was stopped—17¼ pounds
Average gain per week—5.44 ounces
Complementary feeding cases—11
Average gain per week during complemental period—6 ounces
Average complemental period—3.4 weeks
Gastrointestinal upsets—3
Infections: Upper respiratory—6
Otitis media— 4
Pylorospasm—One that cleared on atropine therapy in 10 days

ATHREPTIC GROUP—17 cases

Average birth weight—7 pounds, 15 ounces
Average age when food was started—2½ months
Earliest age started—1¾ months
Oldest age started—6 months
Average age when food was stopped—6.6 months

Oldest age when food was stopped—12.5 months
Average time on food—10 weeks
Shortest period on food—6 weeks
Longest period on food—48 weeks
Average weight when started—9 pounds
Average weight when stopped—16 pounds
Average gain per week—5.4 ounces
Gastrointestinal upsets—1
Infections: Upper respiratory without otitis media—2
Organic causes for athrepsia:
1. Microcephalus
2. Harelip and cleft palate
3. Poliomyelitis
Deaths—2

Vitamin Deficiency.—Vitamin C was supplied in the form of orange or tomato juice and no scurvy was seen in any of the cases.

Of the 117 reportable cases 100 showed no manifestations of rickets when put on the food. Of the group, 52 were given no antirachitic treatment whatever and 80 per cent were protected against rickets, the rest showing some manifestation, such as beading, craniotabes, bossae, epiphyseal enlargement or flaring of the ribs. With the remaining 48 cases cod liver oil and irradiated ergosterol, 3 teaspoonfuls, and 10 drops of 250D, respectively, were used as antirachitic agents. In substantiation of our former findings[47] approximately 92 per cent and 82 per cent of the cod liver oil and irradiated ergosterol groups, respectively, were protected against manifestations of rickets. The study does show, however, that there is no predilection to rickets in babies fed dried milk formula, approximately the same number of cases developing clinical rickets on this food when no antirachitic agent is added as when fresh cow's milk is used alone.

SUMMARY

The results of a critical clinical study of 117 infants fed a modified dried milk are reported. It is shown that this dried milk meets the nutritional requirements of the normal infant, Vitamins A and B are both present in the usual quantities found in fresh milk, and vitamins C and D must necessarily be supplied by antiscorbutic and antirachitic agents. This food is easily digested by the normal infant. Although the general practice is to discontinue a modified dried milk at the usual age of weaning, children do well on it even if continued to a year of age or later.

The gain in weight and the physical development in general in our series of cases compare favorably with groups fed various other foods. The excellent average gain in weight in the athreptic group of infants shows the value of this modified dried milk as a concentrated food in the feeding of marantic infants. There was no greater incidence of infection in this group of cases than in a control group.

With the administration of a sufficient amount of antiscorbutic agent no manifestation of scurvy occurred. The incidence of rickets was not any higher than in infants fed fresh cow's milk or other types of infant foods. The incidence of rickets in the group given no antirachitic agent was no higher than in groups fed other foods with no antirachitic agent.

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INFANT AND MATERNAL MORTALITY IN DENVER

F. P. Gengenbach, M.D.
Denver, Colorado

AT A MEETING of the Denver Public Health Council held in December, 1929, the author was asked to discuss Denver's infant mortality rate.

A hasty investigation had shown that in the Statistical Report of Infant Mortality for 1928 covering 729 cities of the United States as published by the American Child Health Association, Denver, in a group of 25 cities of 250,000 population or over, stood at the bottom of the list with an infant mortality rate of 91 per 1000 live births.

Furthermore, while Denver's rate for 1915 was also 91 as compared to 100 in the United States Registration Area, the rate for the latter had declined to 69 by 1928, while Denver's rate remained the same. This is shown in Table I.

Table I

Infant Mortality Rates

Denver	1915— 91	1928—91
U. S. Reg.	1915—100	1928—69
New York	1917— 89	1928—59
Cleveland	1917—109	1928—60
Minneapolis	1917— 71	1928—51
Omaha	1917— 75	1928—56
Portland	1917— 62	1928—43

In trying to find an explanation for the above figures the first thought was that owing to Colorado's reputation as a health resort for tuberculous patients, perhaps more infants died of tuberculosis in Denver than in other cities. Investigation, however, showed only 7 infant deaths out of 484 had been due to tuberculosis in 1928; and incidentally that the general mortality rate for tuberculosis in Denver from 1915 to 1928 had been cut approximately in half, while the infant mortality rate remained unchanged. The next thought was that again because of our tuberculosis problem many of the mothers might be giving their babies a poorer start in life by not surviving the hazards of childbirth, or by not being able to nurse their babies after they were born.

Investigation showed that while the maternal mortality rate in the United States Registration Area was 6.1 per 1000 live births in 1915, and was still only 6.6 in 1926, Denver's rate was 16.1 in 1915 and 15.8 in 1927, just about two and a half times as high.

Submitted for publication July 8, 1932.

TABLE II

MATERNAL MORTALITY RATES

Denver	1915—16.1	1927—15.8
U. S. Reg.	1915— 6.1	1924— 6.6
Portland	1919—11.8	1926— 6.6
Los Angeles	1919— 8.9	1926— 6.5
	Denver 2½ times as high	

That Denver's record was no worse than the rest of the state was shown by statistics from 1922 to 1926 (Chart I), giving Colorado an infant mortality rate of 92 per 1000 live births, prematurity having been

TABLE III

COLORADO

	1924	1925	1926	1927	1928
Congenital debility, icterus, sclerema	93	72	66	69	59
Premature birth	503	409	383	402	448
Injury at birth	86	68	79	86	95
Other diseases peculiar to infancy	67	69	59	57	61
Lack of care	4	4	3	6	4

given as the greatest cause of death. This is shown in Table III. The rate for the United States Registration Area for the same years was only 75 per 1000 live births as is shown in Chart I.

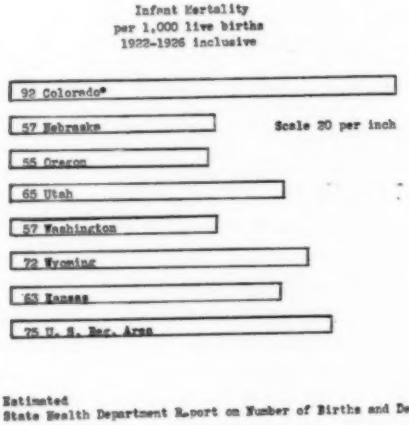

Chart. I.

The presentation of these statistics aroused so much interest in the Council that a committee was appointed, with the author as chairman, to make an intensive survey of all the births in Denver for an entire year, combined with a special study of all of those infants dying within a year of their birth, and also of all maternal deaths occurring during

the survey year. The study included all births occurring in Denver from March 1, 1930, to February 28, 1931; and deaths occurring in this group from the former date to February 28, 1932.*

A very comprehensive questionnaire of some 150 questions was used in the survey, and it is interesting to note that only 11 refusals to answer the questionnaire were recorded and that of these all but two have since complied with the request for information.

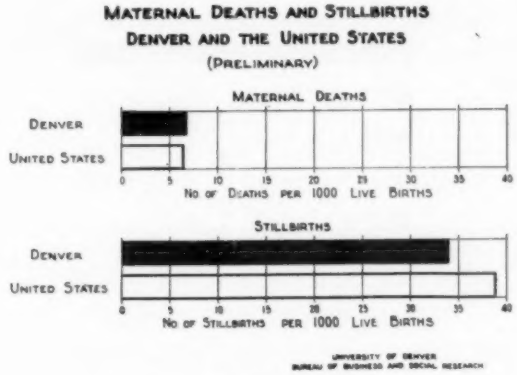
MATERNAL DEATHS AND STILLBIRTHS
DENVER AND THE UNITED STATES
(PRELIMINARY)
MATERNAL DEATHS
DENVER
UNITED STATES
0 5 10 15 20 25 30 35 40
No. of Deaths per 1000 Live Births
STILLBIRTHS
DENVER
UNITED STATES
0 5 10 15 20 25 30 35 40
No. of Stillbirths per 1000 Live Births
UNIVERSITY OF DENVER
BUREAU OF BUSINESS AND SOCIAL RESEARCH

Chart II.

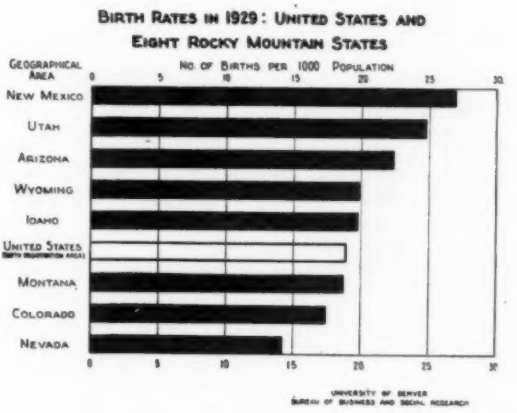

Chart III.

*The field work was done by the Visiting Nurse Association of Denver, in cooperation with the physicians delivering the infants, and the hospitals in which many of the infants were born.

The statistical work was done by Dr. A. D. H. Kaplan, head of the Denver University Bureau of Research, and Professor F. L. Carmichael, head of the University of Denver Bureau of Statistics.

The funds for the survey, about $12,000, were supplied partly from the funds provided for research studies at the University of Denver by the Laura Spelman Rockefeller Foundation, and the other half through the combined efforts of the Denver Health Department, the May Company of Denver, and an anonymous friend.

It may be of interest, too, to note that the questionnaire revealed incidentally that the average number of children to each family is only two in Denver.

Unfortunately, these funds have proved insufficient to provide for a complete study of all of the facts acquired for the whole year, so it

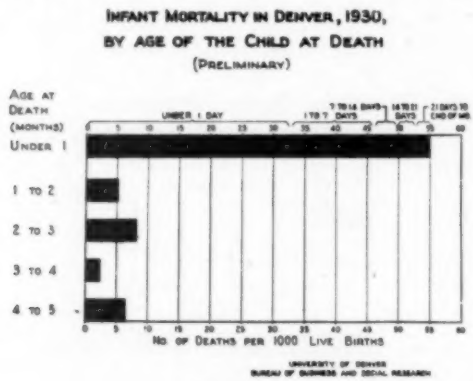
INFANT MORTALITY IN DENVER, 1930,
BY AGE OF THE CHILD AT DEATH
(PRELIMINARY)
AGE AT DEATH (MONTHS)
UNDER 1 DAY
1 TO 7 DAYS
7 TO 14 DAYS
14 TO 21 DAYS
21 DAYS TO END OF MO.
UNDER 1
1 TO 2
2 TO 3
3 TO 4
4 TO 5
0 5 10 15 20 25 30 35 40 45 50 55 60
NO. OF DEATHS PER 1000 LIVE BIRTHS
UNIVERSITY OF DENVER
BUREAU OF BUSINESS AND SOCIAL RESEARCH

Chart IV.

is only possible to give a preliminary report covering some of the chief sociological rather than medical factors determined largely from a study of the first six months of the survey.

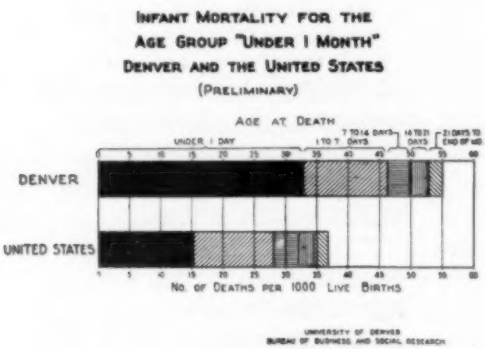
INFANT MORTALITY FOR THE
AGE GROUP "UNDER 1 MONTH"
DENVER AND THE UNITED STATES
(PRELIMINARY)
AGE AT DEATH
UNDER 1 DAY
1 TO 7 DAYS
7 TO 14 DAYS
14 TO 21 DAYS
21 DAYS TO END OF MO.
DENVER
UNITED STATES
0 5 10 15 20 25 30 35 40 45 50 55 60
NO. OF DEATHS PER 1000 LIVE BIRTHS
UNIVERSITY OF DENVER
BUREAU OF BUSINESS AND SOCIAL RESEARCH

Chart V.

There were 5330 births during the survey year, of which however only 4371 were in the families of strictly Denver residents. The other 939 births were in families from outside of Denver, or else had no definite place to live in Denver. Therefore, it is only fair to Denver to point out the fact that it is the only large city in a rather wide territory, sparsely settled, and that many residents of outlying districts come to Denver for emergency treatment, and if death occurs Denver's mortality rates are correspondingly increased.

TABLE IV

BIRTHS AND DEATHS IN DENVER

	BIRTHS	DEATHS	RATE
Total	5330	405	76
Residents	4371	345	79
Nonresidents	959	60	63
SURVEY COVERS			
Births—	March 1, 1930, to Feb. 28, 1931		
Deaths—	March 1, 1930, to Feb. 28, 1932		

Of these 5330 births there were 405 infants who died within a year of their birth, or 76 per 1000 live births, and of the 4371 births there were 345 deaths, or 79 per 1000 live births. The apparent lower rate in nonresidents, 60 deaths in 959 live births, or a rate of about 63, is

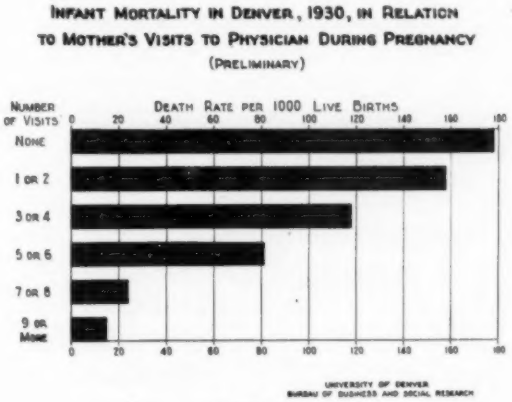

Chart VI.

probably explainable on the basis that all of these infants could not be followed up for a whole year after their birth. During the survey year there were 42 mothers who died in childbirth and from associated causes, giving a maternal rate of 7.88, which is much closer to that of the United States Registration Area. Many of these latter deaths were apparently due largely to lack of proper care. The stillbirth rate for Denver was 38 per 1000 live births, compared with 48 for the United States Registration Area.

INFLUENCE OF NEONATAL MORTALITY

Infant mortality rate by age of the child is shown in Chart IV. There were 33 deaths under one day; 46 the first week; 50 within two weeks; 53 within three weeks; and 55 within the first month after birth. The rate between one and two months was 5; between two and three months 8; between three and four months 2.5; and between four and five months 6.5 per 1000 live births.

Statistics for the United States Registration Area under one month showed 15 under one day; 28 within one week; 32 within two weeks; 34.5 within three weeks; and 37 the first month. A comparison of Denver with the United States Registration Area is shown in Chart V.

VALUE OF PRENATAL CARE

The apparent value of prenatal care was evidenced by the finding that where the mother had made no visits to the physician before the infant was born the rate was 178; in the case of 1 or 2 visits the rate was 158; with 3 or 4 visits 118; with 5 or 6 visits 80; with 7 or 8 visits 22; and with 9 or more visits to the physician before labor occurred 15 per 1000 live births. These data are shown in Chart VI.

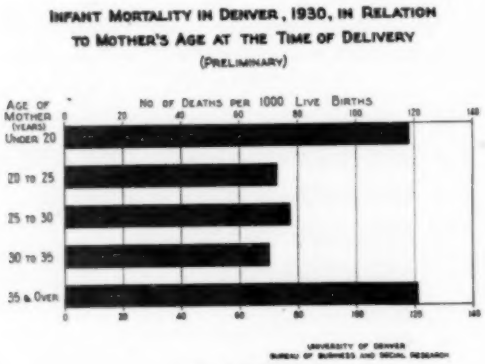

Chart VII.

INFLUENCE OF MOTHER'S AGE

The relation of the mother's age at the time of delivery to the infant mortality rate is shown in Chart VII. In the case of mothers under twenty years the rate was about 118; between twenty and twenty-five years 74; between twenty-five and thirty years 78; between thirty and thirty-five years 70; and thirty-five years and over 121 per 1000 live births.

INFLUENCE OF STANDARD OF LIVING

In relation to the family's annual income for those under $500 the rate was about 168; between $500 and $1000 was 106; between $1000 and $1500 was 96; between $1500 and $2000 was 56; between $2000 and $2500 was 54; between $2500 and $3000 was 38; and for $3000 and over was 30 per 1000 live births. This is shown graphically in Chart VIII.

Comparing births and deaths during the first six months of the survey with annual income we found under $500 the percentage of births was—5.1, and deaths—11.1; $500 to $1000, births—17.9, and deaths—

24.2; $1000 to $1500, births—29.5, and deaths—35.9; $1500 to $2000, births—20.2, and deaths—14.4; $2000 to $2500, births—10.5, and deaths—7.2; $2500 to $3000, births—8.2, and deaths—3.9; and $3000 and over was births—8.6, and deaths—3.3 per 1000 live births.

TABLE V

BIRTHS AND DEATHS FIRST SIX MONTHS BY ANNUAL INCOME

Under $500	5.1 l.b.	11.1 deaths
$500 to $1000	17.9 l.b.	24.2 deaths
$1000 to $1500	29.5 l.b.	35.9 deaths
$1500 to $2000	20.2 l.b.	14.4 deaths
$2000 to $2500	10.5 l.b.	7.2 deaths
$2500 to $3000	8.2 l.b.	3.9 deaths
$3000 and over	8.6 l.b.	3.3 deaths
	100.0	100.0

These findings as shown in Table V would indicate that not until the family income reaches about $150 a month can the family begin to afford adequate medical and nursing care for the pregnant mother.

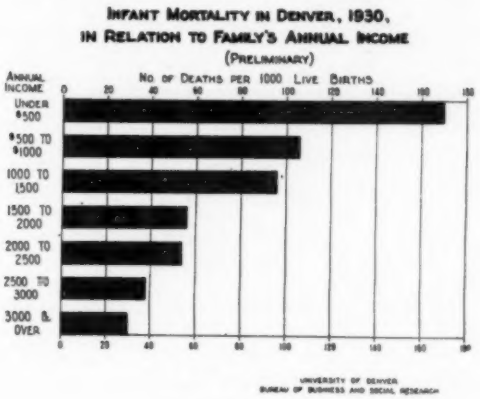

Chart VIII.

INFLUENCE OF FOREIGN POPULATION

The relation of the presence of a foreign population with inadequate family income is well shown by our findings in connection with our Mexican population, which is only 2 per cent of the total population of

TABLE VI

MEXICAN INFANT MORTALITY IN DENVER

Population	is 2.0% of total
Live births	is 5.5% of total
Deaths under 1 year	is 12.2% of total
Denver infant death rate	86 per 1000 live births
Mexican death rate	193 per 1000 live births

Denver. As shown in Table VI the Mexican births were 5.5 per cent of the total, and the deaths under one year were 12.2 per cent of the total infant mortality in Denver. So that while Denver's infant mortality rate during the first six months of the survey was 86 per 1000 live births, the Mexican rate was 193 per 1000 live births, or more than double.

SECTIONS OF THE CITY

A graph of the various sections of Denver showed a so-called "black belt" of highest infant mortality along the South Platte River bottoms, where many of the Mexicans live, and where the standard of living is very low. (See Chart IX.)

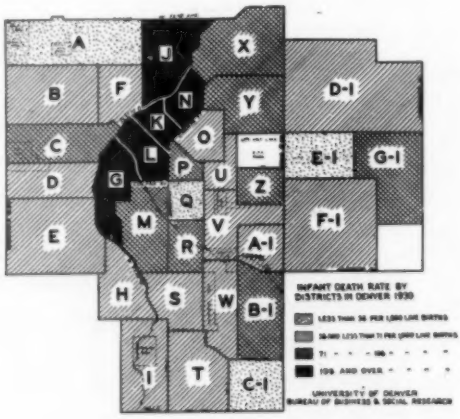

Chart IX.

POSSIBLE VALUE OF EDUCATIONAL PROPAGANDA

Whether it will only prove to be a coincidence or not, as shown by infant mortality rates in future years in Denver, yet it is interesting to note the following figures for 1930 and 1931, as showing the possible value of the educational propaganda brought about by the survey.

TABLE VII

BIRTHS AND DEATHS IN DENVER, COLORADO
(Rate per 1000 Live Births)

	1930	1931
Births	5273	4702
Deaths	496	356
Stillbirths	175	143
Rate	94	76

In 1930 there were 5273 births and 496 deaths under one year, or an infant mortality rate of 94 per 1000 live births, and in 1931 there were 4702 births and 356 deaths under one year, or an infant mortality rate of 76 per 1000 live births.

CONVALESCENT SERUM IN THE PREVENTION OF MUMPS

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EPIDEMICS of mumps in an institution containing many susceptible persons are difficult to control. Due to the long incubation period of the disease these epidemics may continue for months with new cases developing at long intervals.

Although mumps is usually a mild infection in small children, orchitis is not an infrequent complication in boys past the age of puberty, and there are other obvious reasons which make it desirable to protect susceptible persons if it is possible.

On Jan. 31, 1932, the first case of mumps appeared in the Glenview School, a boarding school for boys. The following day a second boy became ill with the disease. On Feb. 14 the third case developed, followed the next day by another. Other cases developed on Feb. 19, 23, and 29. The boys suffering from the disease were sent to the contagious wards of the Cincinnati General Hospital as soon as a diagnosis was made. The disease was of moderate severity and uncomplicated. It was bilateral in most instances and the swelling of the parotids disappeared in from six to eighteen days, the average being twelve days.

On March 4, blood was taken from the six of the boys who had just recovered from the disease. This was from six to thirty-two days after the appearance of the first symptom and one to seventeen days after the disappearance of the parotid swelling. Serum was prepared from this blood by Dr. Merlin L. Cooper of the Children's Hospital Research Foundation. It was not pooled, each boy's blood being kept separate. The Wassermann test on each specimen was negative.

On March 7, seven days after the last exposure, 5 c.c. of this convalescent's serum was injected intramuscularly into 44 boys who, according to the history obtained from the parents, had not had mumps. They varied in age from ten to eighteen years. They had been definitely exposed, since they slept in dormitories with the beds quite close together, ate in common dining rooms and were together in the classrooms and playgrounds. At the time of this report more than three months after the injection, no more cases of mumps have appeared.

Alfred Hess[1] in 1915 was apparently the first to employ blood from patients convalescent from mumps for the prevention of the disease. He used intramuscular injections of 6 to 8 c.c. of whole blood. Seventeen susceptible children so treated did not develop the disease while

From the B. K. Rachford Department of Pediatrics, College of Medicine, University of Cincinnati. Aided by the Children's Hospital Research Foundation.
Submitted for publication, June 6, 1932.

37 per cent of 117 other susceptible children not injected, developed it. In 1925 Regan[2] injected 2 to 4 c.c. of convalescent's mumps serum into each of 70 children before the seventh day following exposure, and only one developed the disease. In 1927 Skrotsky[3] employed 5 to 15 c.c. of convalescent's serum in each of 179 children who had been definitely exposed to patients suffering from mumps and only two developed the disease. Barenberg and Ostroff[4] gave 12 c.c. of whole blood from patients convalescent from mumps to 55 susceptible children. Eight, or 15 per cent, developed the disease in contrast to 49, or 39 per cent, of 125 children who did not receive the treatment. In the 8 injected children there was only one severe case, while among the 49 untreated there were 28 severe cases.

SUMMARY

Forty-four boys ranging in age from ten to eighteen years, who according to their history had never had mumps, were injected with 5 c.c. of convalescent's serum seven days after exposure to the disease. None developed mumps.

It would appear from the above report and from the data in the literature that convalescent's serum is of definite value in the prevention of mumps in exposed persons if employed early and in sufficient dosage.

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670 Rockdale Avenue.

INFLUENCE OF THE AGE FACTOR ON THE FREQUENCY AND MONTHLY SEASONAL INCIDENCE OF RESPIRATORY INFECTIONS

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Birmingham, Alabama

TOWNSEND[1] in a review of the literature on respiratory infections states, "There has been little advance in any knowledge concerning its etiology, epidemiology, periodicity or its diagnosis." Barrow[2] suggests, "That any study of possible factors governing the susceptibility of respiratory infections is worth reporting, since even negative conclusions help to narrow the field of investigations." Sydenstricker[3] of the United States Public Health Service found that more than half of the total deaths of all ages were due to respiratory diseases, notwithstanding the fact that the ratio of respiratory illnesses to deaths from respiratory causes was 300 to 1. These figures are almost identical with the statistics of the United States Army.[4] In Chicago, in 1930, of 3,102 deaths occurring in children under one year of age, respiratory infections were responsible for 35 per cent of the total, premature births, birth injuries, and congenital malformations not included. In a four-year observation of a private school in New York City attended by children between the ages of six and eighteen years, Mason[5] found that of the total number of days lost from school as the result of illness, respiratory infections were responsible for 64 per cent; contagious diseases 23 per cent; and all other illness 13 per cent. The 23 per cent of days lost from school due to contagious diseases includes the time of quarantine of children having the disease as well as the number of days lost from school in a certain number of contact children who were quarantined but did not develop the disease. Statistics compiled by Smith,[6] Sanford,[7] and Lincoln[8] show that respiratory infections were responsible for approximately the same percentage of days' absence from school due to illness as those of Mason.

Respiratory disease, one of the important economic and social problems of the day, probably comprises approximately 75 per cent of the total cost to the wage earner for medical service for himself and family and for the time lost from work due to acute medical illness.

In the hope that some light might possibly be thrown on the future solution of this problem,[9] observations and investigations of respiratory infections, regardless of positive or negative findings, are worth consideration.

Submitted for publication, July 11, 1932.

The following analysis of 1,255 respiratory infections was made in an endeavor to find out if the age of children has any apparent effect on the number and seasonal incidence of the infections.

The 1,255 respiratory infections were observed in 156 children during an exact period of four years from the date of their birth. The infections occurred over a ten-year period and not in the same calendar years. The children included in the series are patients with whom the writer has been in intimate contact and feels reasonably sure were seen in practically all illnesses. No infections are reported except those in which the patients were seen professionally. Eighty-three, or 53 per cent, of the children were under observation before the age of two months. One hundred eighteen, or 76 per cent, were seen before the age of four months.

In eighty-four children respiratory infections were first seen during the first six months of life; in 61 during the second six months; and in 3 at the age of twelve months. Eight children apparently had no respiratory infections until after the twelfth month of age. Of these, 7 were under observation before the age of six months, and 1 at the age of ten months. Apparently there was no consistency in either the age or the month of the first infection.

During the first four years of life, approximately 40 per cent of the 1,255 respiratory infections were mild and 60 per cent severe. The mild infections included ambulatory cases, with little or no fever, and were made up principally of acute upper respiratory types of infections. The severe infections included bed cases acutely ill, with high temperature, and were composed principally of so-called influenza with its various complications: acute bronchitis, pneumonia, acute follicular tonsillitis, etc.

TABLE I

THE YEARLY INCIDENCE OF 1,255 RESPIRATORY INFECTIONS OCCURRING IN 156 CHILDREN DURING AN EXACT PERIOD OF FOUR YEARS FROM DATE OF BIRTH, WITH GROUPING ACCORDING TO MONTH OF BIRTH

MONTH OF BIRTH	NO. PATIENTS	NUMBER OF RESPIRATORY INFECTIONS PER YEAR					'VER. NO. INF. PER PT.
		1ST YR.	2ND YR.	3RD YR.	4TH YR.	TOTAL	
Sept.	12	25	33	22	20	100	8.33
Oct.	14	21	38	21	32	112	8.
Nov.	9	15	18	22	15	70	7.77
Dec.	8	13	22	16	17	68	8.5
Jan.	10	19	28	21	22	90	9.
Feb.	13	32	27	27	20	106	8.14
Mar.	9	20	20	15	21	76	8.33
Apr.	17	35	44	36	32	147	8.64
May	19	39	38	34	29	140	7.84
June	19	35	40	40	34	149	7.9
July	12	19	32	19	12	82	6.83
Aug.	14	30	36	27	22	115	8.21
Total	156	303	376	300	276	1,255	8.04

Among the children are included a few patients with so-called allergic manifestations. This type of patient almost invariably showed signs and symptoms suggestive of a more or less chronic paranasal sinusitis. No infections are included in this group except those in which there was definite evidence of an acute respiratory infection.

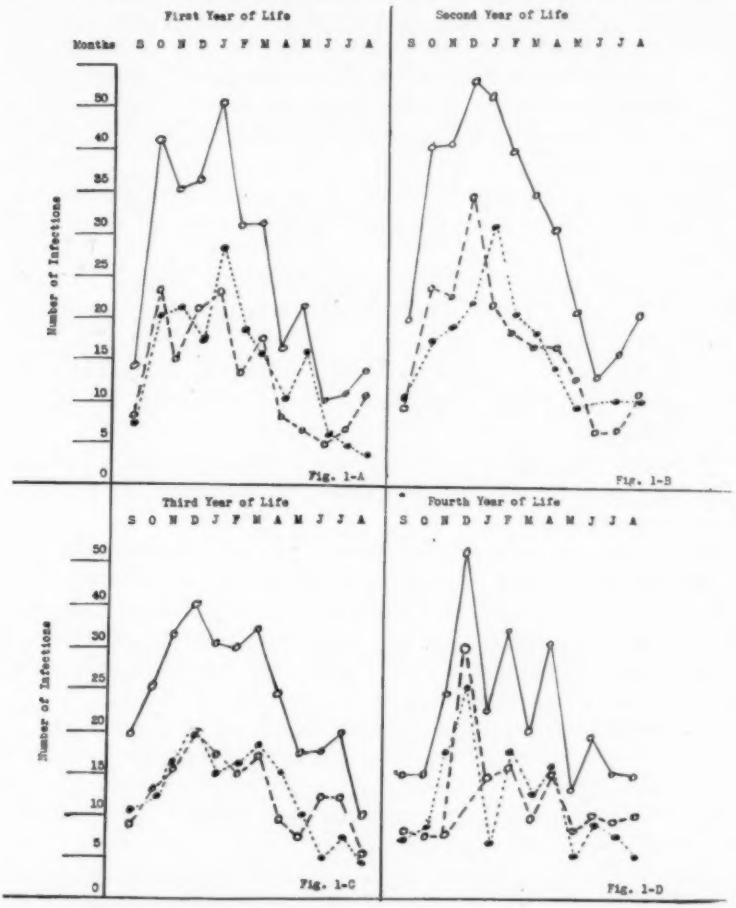

Fig. 1.—The monthly seasonal incidence of 1,255 respiratory infections as seen during the first, second, third and fourth years of life in 156 children, divided into two groups of 78 cases each, using alternate patients, according to month of birth.

Fig. 1-A—1st year. Total infections, 303; Group I, 147; Group II, 156; difference, 9 infections.

Fig. 1-B—2nd year. Total infections, 376; Group I, 194; Group II, 182; difference, 12 infections.

Fig. 1-C—3rd year. Total infections, 300; Group I, 152; Group II, 148; difference, 4 infections.

Fig. 1-D—4th year. Total infections, 276; Group I, 142; Group II, 134; difference, 8 infections.

First 4 years. Total infections, 1255; Group I, 635; Group II, 620; difference, 15 infections.

Solid line; total infections; broken line, Group I; dotted line, Group II.

In Table I, the patients are grouped according to the month of birth, and the number of respiratory infections during the first, second, third, and fourth years of life are tabulated.

Fig. 1 is compiled from the patients included in Table I. The 156 children are divided into two groups of 78, using alternate patients according to month of birth. The graph shows the monthly seasonal incidence of the total number, and the number of infections occurring in the two groups during the first, second, third, and fourth years of life.

In the first year of life 303 infections were seen; one group having 147 and the other 156, a difference of 9 infections between the two groups (Fig. 1-A).

In the second year of life 376 infections were seen; one group having 194 and the other 182, a difference of 12 infections between the two groups (Fig. 1-B).

In the third year of life 300 infections were seen; one group having 152 and the other 148, a difference of 4 infections between the two groups (Fig. 1-C).

In the fourth year of life 276 infections were seen; one group having 142 and the other 134, a difference of 8 infections between the two groups (Fig. 1-D).

During the first four years of life in the 156 children, 1,255 respiratory infections were seen; Group I having 635 and Group II, 620, a difference of 15 infections.

In the first two years of life Group I had 341 infections and Group II had 338, a difference of 3 infections between the two groups.

In the second two years of life Group I had 294 infections and Group II had 282, a difference of 12 infections between the two groups.

There was a difference of 59 infections between the first two years and the second two years of life in Groups I and II.

The analysis of the monthly seasonal incidence of respiratory infections in the two groups of children during the first, second, and especially the third and fourth years of life, shows evidence that the age of the child apparently influenced the monthly seasonal incidence of the infections.

The effect of an epidemic year of respiratory infections on the number of infections seen in individual patients is shown in Fig. 2. During the fall and winter of 1928-29 occurred the largest epidemic of respiratory infections seen in Birmingham, Ala., during the past ten years.

From Sept. 1, 1928, to June 1, 1929, 933 respiratory infections were seen in 582 patients. Fig. 2-A is a graph of the number of infections seen each calendar month during this period.

The peak in the epidemic was the month of December when 286, or 30 per cent of the total number of infections, were seen.

Fig. 2-B shows the number of respiratory infections seen in 28 of the 156 children reported during the years 1927-1928 and 1929-1930 compared with the epidemic year 1928-1929.

From Sept. 1, 1927, to Sept. 1, 1928, the year preceding the epidemic, 78 infections were seen in 28 children. From Sept. 1, 1928, to Sept. 1, 1929, the epidemic year, the 28 children were seen with 64 respiratory

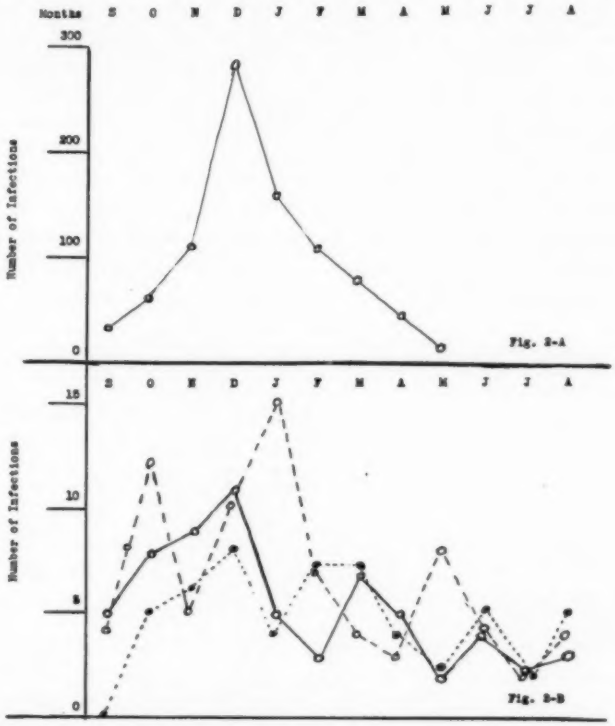

Fig. 2.—Effect of an epidemic year of respiratory infections on the number and seasonal incidence of infections seen in individual patients.

Fig. 2-A The monthly seasonal incidence of 933 respiratory infections seen in 582 children from September 1, 1928 to June 1, 1929. (Epidemic year.)

Fig. 2-B The number and the monthly seasonal incidence of respiratory infections seen each year in 28 of the 156 children reported from September 1, 1927 to September 1, 1930.

Broken line 1927-1928; 78 respiratory infections.
Solid line 1928-1929; 64 respiratory infections. (Epidemic year.)
Dotted line 1929-1930; 53 respiratory infections.

infections. From Sept. 1, 1929, to Sept. 1, 1930, the year following the epidemic, the 28 children were seen with 53 infections.

The 28 children in 1927-1928 had 14 more infections than in 1928-1929, the epidemic year, and 25 more infections than in 1929-1930, the year following the epidemic.

TABLE II

ONE HUNDRED FIFTY-SIX CHILDREN OBSERVED FOR AN EXACT PERIOD OF FOUR YEARS FROM DATE OF BIRTH ARE PLACED IN TWO GROUPS OF 78 PATIENTS AND THE NUMBER OF RESPIRATORY INFECTIONS SEEN DURING THE FIRST, SECOND, THIRD AND FOURTH YEARS OF LIFE IN THE TWO GROUPS ARE COMPARED

	NO. PATIENTS	NUMBER OF RESPIRATORY INFECTIONS THE FIRST FOUR YEARS OF LIFE				
		1ST YR.	2ND YR.	3RD YR.	4TH YR.	TOTAL
Group I	78	147	194	152	142	635
Group II	78	156	182	148	134	620
Diff. in No. R. I.		9	12	4	8	15

The number of infections and the monthly seasonal incidence apparently depended upon the age of the children, rather than on the epidemic. (Table III and Fig. 1.)

In 1927-1928 the 28 children were younger than two years of age. Table III shows that in normal years children have more respiratory infections during the first two than in the second two years of life and the largest number of infections occurred in the month of January. (Fig. 1-A and Fig. 1-B.)

TABLE III

ONE HUNDRED FIFTY-SIX CHILDREN OBSERVED FOR AN EXACT PERIOD OF FOUR YEARS FROM DATE OF BIRTH ARE PLACED IN TWO GROUPS OF 78 PATIENTS AND THE NUMBER OF RESPIRATORY INFECTIONS SEEN THE FIRST TWO YEARS OF LIFE ARE COMPARED WITH THE NUMBER SEEN THE SECOND TWO YEARS OF LIFE

	NO. PATIENTS	NUMBER OF RESPIRATORY INFECTIONS THE FIRST FOUR YEARS OF LIFE		
		NO. 1ST TWO YR.	NO. 2ND TWO YR.	DIFF. IN NO. BETWEEN 1ST AND 2ND TWO YR.
Group I	78	341	294	47 (59)
Group II	78	338	282	56 (44)
Diff. in No. R. I.		3	12	

In 1928-1929, the epidemic year, the 28 children were older, having 14 fewer infections than in the preceding year, corresponding to the number of infections seen in normal years in children of the same age. (Table III.) During the epidemic year, 1928-1929, the monthly peak in the number of infections was in December, the same as in the epidemic, but this peak is no greater than in 1929-1930 or in normal years in children of the same age, as is shown in Fig. 1-C and Fig. 1-D.

COMMENT

It has been shown by various investigators that the severity and the peaks in the monthly seasonal incidence of respiratory infections are influenced largely by climatic conditions and geographic locations.

Townsend,[1] Smiley,[10] and Barrow,[2] found that climatic conditions, geographic location, living and personal hygiene of individuals, apparently had little effect on their susceptibility to repeated attacks of respiratory infections.

McLean's[11, 12, 13] observations have shown that apparently weather conditions do not influence the number of respiratory infections. Recurrent respiratory infections are common in children of all ages and occurred each year in about the same percentage of cases. The removal of tonsils and adenoids does not influence either the number or severity of respiratory infections.[13] There is a remarkable consistency in the sameness of the total number, average number of infections per patient, and the average length of time between respiratory infections in various groupings of children of the same age, number and length of observation. This consistency is not found when children of different ages are compared.

During a 10 calendar year period in which 156 children were observed for an exact period of four years from the date of their birth, 1,255 respiratory infections were seen. The cases were placed in two groups of 78 patients each and the number of respiratory infections seen in the two groups during the first, second, third, and fourth years of life, when analyzed, showed evidence that the age of the child influenced both the number and the monthly seasonal incidence of the infections.

In 28 of the 156 children during an epidemic year of respiratory infections the number and the peaks in the monthly seasonal incidence of the infections were apparently influenced more by the age of the child than by the epidemic.

SUMMARY

In individual patients during an epidemic year of respiratory infections, apparently the age factor and not the epidemic, influenced the number and the peaks in the monthly seasonal incidence of the infections.

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ARGYRIA

Report of a Case in a Patient Aged Five and a Half Years

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ON SEPTEMBER 7, 1931, G. L., female, aged five and a half years, was admitted to the University of Virginia Hospital with a statement from her mother that the child was suffering from heart disease and had been so diagnosed by her family physician.

Past History.—Said to have been a normal baby at birth. The delivery was said to have been normal, and she got along well after birth and through the first year of life. At the age of one year, she had influenza followed by otitis media and mastoiditis on the right side and was operated on for the latter condition. Every winter since then she has had to have the left ear drum punctured. She is subject to frequent colds and a nasal discharge, and her rhinologist has told the mother that she had a nasal trouble. The mother states that at the time of the mastoid operation she was told that the child had a brain abscess. About three years ago a second doctor told the mother that her child must have heart trouble because of the peculiar color of her skin which developed between the operation on the mastoid and the time of this note. The mother also noticed that the child had a bluish tinge to the skin. The family physician did not think at first that she had heart trouble but is said to think so now. Patient is usually not short of breath though mother states that this occurs after extreme exertion. She has never had any edema of the feet, ankles or face. Her color is somewhat bluer after exertion and often during her bath. The child has not taken any drugs that the mother knows of such as aspirin or acetanilid, but the mother states that several years ago the nose was irrigated frequently with large amounts of a solution of neosilvol. Mother "guesses a gallon was used in the nose" (it is not unlikely that the child swallowed considerable amounts of the solution). The patient has had chickenpox and whooping cough both with good recovery. There is no history of measles, mumps, scarlet fever, diphtheria or rheumatic fever. Tonsils have been removed. Family history is essentially negative.

Physical Examination.—Aged five years and six months, height 47¼ inches, weight 38 pounds. (Normal for height is 50 pounds, 8 ounces.) The general appearance of the child is that of a somewhat underdeveloped and not particularly well nourished child. The lungs are negative on percussion and no râles are heard. The breath sounds at the left apex are thought to be a little harsher than normal. *Neck:* Negative except that the thyroid is palpable, smooth and symmetrical. *Throat:* Tonsils have been removed. Posterior pharyngeal wall slightly reddened and follicular, and there is an evident postnasal discharge. *Teeth:* Deciduous teeth, somewhat carious. Permanent teeth are erupting in front. *Heart:* Soft systolic murmur heard all over the precordium and to the apex, not transmitted toward the axilla, no diastolic murmur, no evidence of enlargement on percussion. Sinus arrhythmia. *Skin:* Bluish tinge all over body including finger nails, though this is slight and there is no club-

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bing of fingers. Mucous membrane of the lips is slightly blue and that of the mouth decidedly blue; this is especially noticeable on the hard palate which shows a metallic sheen. It is noteworthy that when the child is put in a supine position and the head allowed to hang over the edge of the table the lips become somewhat redder as does the mucous membrane over the hard palate, which loses the metallic glisten.

Progress While in Hospital.—Patient remained in hospital five days without noticeable change in her condition except that the murmur over the heart tended to disappear and return, in other words, was exceedingly inconstant and transient. With exercise it disappeared completely. The temperature remained between 98° and 99° and the pulse remained constantly below 100. The respiration averaged 20.

Laboratory Findings.—Urinalysis: Amount small, color—dark yellow and cloudy, reaction acid, specific gravity 1.006, albumin none, sugar none. Microscopic: White blood cells, 20 to 25; red blood cells—an occasional one found; a few epithelial cells and no casts. Urinalysis was repeated several times and what has just been stated was an average finding. *Blood:* Hemoglobin 59 per cent (Sahli), equal 38 gm. per 100 c.c.; red blood cells 4,490,000; white blood cells 5,800; polymorphonuclears 56 (bards 5, segments 51); lymphocytes 30; large mononuclears 7; transitionals 3; eosinophils 4. No anisocytosis, poikilocytosis, or polychromatophilia. No nucleated red blood cells, no parasites. Platelets not counted but apparently normal.

Stool Examination.—Color—dark brown, formed; no gross mucus, no gross blood; benzidine test negative; no ova, pus, or parasites. Fat not increased, starch moderate in amount.

Wassermann.—Blood was taken for a Wassermann but was lost by accident and the patient left the hospital before it could be taken again. Oxygen content of venous blood 6.5 vol. per cent.

X-ray Examination.—September 7, 1931. Screen examination of the chest negative. On September 8, 1931: a seven-foot film was taken with the following measurements:

A	3.0 cm.
M. R.	2.5 cm.
M. L.	6.0 cm.
Bony thorax	16.0 cm.
Percentage	53 per cent

The measurements are thought to be practically within the limits of normal for a child of this age. No abnormalities of contour were demonstrated.

On September 11, 1931, an electrocardiogram was made with the following report:

Rate and rhythm—Normal rhythm; rate 120.

PR interval—0.16.

QRS complexes—Normal.

T-waves—Upright in Lead I, flattened in Lead II, flattened and inverted in Lead III.

Axis deviation—None (+12).

Conclusion: Normal tracing.

Biopsy of Skin.—On September eleventh a biopsy of the skin was made. A section of skin 3 × 1½ × ¾ cm. was removed which showed a rather bronze color. Microscopically the skin was normal except for a brown pigment distributed in the basal layer of the sweat glands. Also some streaks of pigment seen about the sweat glands. In the basal layer of the epidermis there is some pigment which may

be normal pigment. While this deposit of pigment may be due to either silver or bismuth the latter can be eliminated clinically and from the history. *Diagnosis:* Argyria.

While the child was in the hospital it was decided that the heart was normal and that the transient and inconstant murmur was entirely functional. The conclusion was reached that the condition was one of Argyria.

NOTE.—A letter just received from this child's mother and her present attending physician indicates that the color is increasing.

THE SEASONAL INCIDENCE OF POLIOMYELITIS IN AND ABOUT CLEVELAND

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ONE hundred thirty-nine cases of infantile paralysis were studied outside and 352 cases inside the Contagious Division of Cleveland City Hospital in the decade from 1921 to 1930. Notes were taken of seasonal conditions and official weather statistics were consulted. In the following paragraphs, we attempt to correlate these notes with the official data.

In our consideration of the subject, it was thought best to plot the seasonal occurrence and to note if there was any effect of temperature, rainfall, precipitation, and sunshine upon the morbidity rate of this disease for Cleveland and vicinity.

SEASONAL OCCURRENCE

During this ten-year period, only 8 cases, a little over 1 per cent of the total number studied, were admitted to Cleveland City Hospital during the winter time. One half of the latter occurred in December and could quite possibly represent cases coming at the extreme end of the epidemic season. None of the 139 patients seen outside the hospital were endemic cases.

During the same years (1921 to 1930 inclusive), the Cleveland Board of Health reported 541 cases of infantile paralysis as shown in Table II, 40 (7+ per cent) having been reported during the winter months.

Some of the hospital winter cases might well have been irritative types of cerebrospinal diseases, such as encephalitis, specific or nonspecific, associated with infectious or contagious diseases, and because of this, the charts of the 8 winter poliomyelitis patients were carefully reexamined. Four were unquestionably infantile paralysis cases, while the other 4 were possibly cases of this type. As was previously noted, 4 of the 8 sporadic cases occurred in December. The remainder were admitted to the hospital at odd intervals from January to June. Of the latter 4 cases, only 2 were unquestionably cases of infantile paralysis. If after a hospital study with all the laboratory means available, one could not be sure of the classification of these aberrant cases, how much less could one be sure of reports coming from unchecked sources where encephalitis and meningitis are commonly mistaken for infantile paralysis and vice versa?

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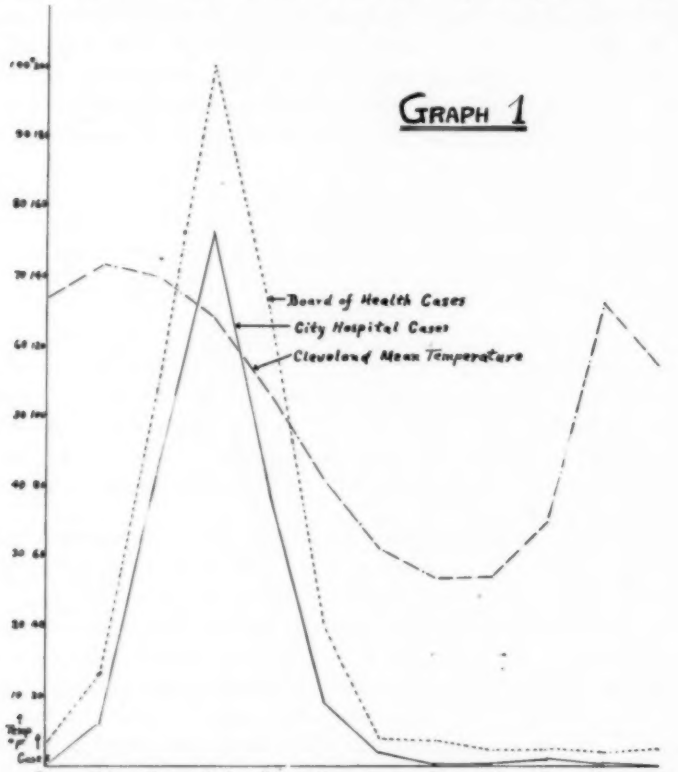
One must be skeptical of a diagnosis of poliomyelitis occurring any time between January and June in this city, even though Wickman[1] has shown that it is not impossible for such an infection to occur during the winter months in other countries and endemic winter cases have been described by competent observers in other parts of the United States.

Based upon our own figures and those of the local Board of Health, a curve of incidence of disease for this locality was plotted; it shows



Seasonal Incidence of Poliomyelitis in Cleveland 1921-1930.

that the height of the incidence peak occurred during the months of August, September, and October (Graph I). Even with endemic cases reported by the physicians included, the precipitous rise of the morbidity curve conclusively demonstrates the seasonal incidence of the disease in this locality.

TEMPERATURE AND SEASONAL INCIDENCE OF POLIOMYELITIS

We have studied the temperature curves for 1921 to 1930 inclusive and cannot find any relationship between mean temperature curves

and a frequency curve of poliomyelitis for the same period. Height of temperature is likewise not of much significance. The mean temperature for ten years is plotted on Graph I and illustrates this point. The average mean temperature is lower in September than in July or August and yet the number of poliomyelitis cases is higher during this month.

Even when the figures are studied more intimately, there is no striking individual monthly variation in temperature indicative of more than a casual relationship. If we take June as an example, we see that in June of 1925, the year of the greatest morbidity rate between 1921 and

TABLE I

SEASONAL PREVALENCE OF INFANTILE PARALYSIS AS SHOWN BY 352 CASES IN CLEVELAND CITY HOSPITAL, INCLUDING 8 ENDEMIC CASES 1921 TO 1930 INCLUSIVE

YEAR	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	TOTAL NO. CASES PER YEAR
1921	0	2	4	3	0	9
1922	0	0	2	0	0	2
1923	0	0	2	1	0	3
1924	2	0	1	5	0	8
1925	1	20	21	12	0	54
1926	0	10	14	9	0	33
1927	0	14	24	10	3	51
1928	5	18	21	4	3	51
1929	4	10	6	4	2	26
1930	1	12	58	26	10	107

Endemic Cases

YEAR	DECEMBER	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	TOTAL NO. CASES PER YEAR
1921	0	0	1?	0	0	0	0	1
1922	0	0	0	0	0	0	0	0
1923	0	0	0	0	0	0	0	0
1924	0	0	0	0	0	0	0	0
1925	0	0	0	1?	0	0	0	1
1926	1?	0	0	0	1	0	0	2
1927	1?	0	0	1	0	0	0	2
1928	0	0	0	0	0	0	0	0
1929	0	0	0	0	0	0	0	0
1930	2	0	0	0	0	0	0	2

1930, the average temperature was 71.6° F., or 7.3° higher than the comparable month in 1926, 8.4° higher in 1927 and 7.6° higher than in 1928 (Table III), although the latter years could also be considered epidemic years. If June high temperatures were of any significance, it would be hard to explain the relative infrequency of poliomyelitis in 1923 (temperature 70.8° F.) or, conversely, why the disease was so prevalent during the years 1926, 1927, and 1928 when the average June temperature curves were lower. Even with the local morbidity increase in 1930, the June temperature of that year was 0.6° below that of 1923, a nonepidemic year. When we consider the other months as

TABLE II

SEASONAL PREVALENCE OF INFANTILE PARALYSIS AS SHOWN BY 541 CASES IN CLEVELAND BOARD OF HEALTH SERIES, INCLUDING 40 ENDEMIC CASES 1921 TO 1930 INCLUSIVE

YEAR	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	TOTAL NO. CASES PER YEAR
1921	9	12	3	5	2	31
1922	0	0	2	3	1	6
1923	0	2	4	3	0	9
1924	2	3	6	8	4	23
1925	3	26	31	18	4	82
1926	1	3	8	7	2	21
1927	0	17	35	10	2	64
1928	8	21	25	8	7	69
1929	2	10	5	4	3	24
1930	2	13	82	64	11	172

Endemic Cases

YEAR	DECEMBER	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	TOTAL NO. CASES PER YEAR
1921	0	0	0	0	0	3	2	5
1922	0	0	1	0	0	0	0	1
1923	1	2	0	1	0	0	0	4
1924	0	0	0	0	0	1	0	1
1925	1	1	0	1	0	0	2	5
1926	2	1	0	0	1	0	0	4
1927	1	2	2	0	0	0	0	5
1928	0	0	2	2	0	0	0	4
1929	1	1	0	1	2	1	2	8
1930	2	0	0	0	1	0	0	3

we have considered the month of June, we find that in no way do the curves for individual months, combination of months, or years, give any evidence that temperature changes in themselves might have any effect on the morbidity rate of this disease.

TABLE III

MONTHLY AND ANNUAL MEAN FAHRENHEIT TEMPERATURES FOR CLEVELAND 1921 TO 1930 INCLUSIVE

YEAR	JAN.	FEB.	MARCH	APRIL	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.	ANNUAL AVER.
1921	32.0	32.1	45.6	53.9	59.8	69.8	76.8	69.3	69.0	53.6	42.0	32.6	53.0
1922	25.6	31.7	38.8	48.6	61.3	68.6	71.6	69.6	67.0	55.4	44.2	31.8	51.2
1923	30.2	23.8	34.8	45.8	54.5	70.8	71.2	69.0	64.7	52.2	42.0	40.4	50.0
1924	24.0	25.4	33.0	45.3	52.4	64.8	69.2	70.0	60.4	55.6	41.2	27.6	47.4
1925	25.6	33.8	39.0	50.6	53.6	71.6	70.8	70.9	67.2	45.2	39.6	28.9	49.7
1926	27.2	28.1	30.8	41.5	56.9	64.3	71.6	72.2	65.1	52.2	40.0	28.0	48.2
1927	26.6	33.7	40.3	47.2	57.2	63.2	71.6	65.4	67.6	57.6	46.6	32.2	50.8
1928	28.4	29.2	35.4	44.2	57.0	64.0	73.1	72.8	61.6	57.4	44.2	35.2	50.2
1929	25.0	25.2	43.6	50.6	56.4	65.8	72.5	68.0	65.6	52.2	39.4	32.0	49.7
1930	27.6	37.5	35.7	48.3	61.8	70.2	73.6	71.1	67.2	51.8	44.8	32.0	51.8
Mean Aver. 1871-1930	26.8	27.1	35.3	46.3	57.8	67.2	71.8	70.0	64.3	53.2	40.9	31.0	49.3

RAINFALL, PRECIPITATION, AND SEASONAL INCIDENCE OF POLIOMYELITIS

Hull[2] studied the records of 34 epidemics reported in different parts of the world during the seventy-five years preceding 1917. Only 18 of these epidemics were sufficiently well reported so that the number of cases by months could be determined. Practically 90 per cent of the 13,365 cases occurred during deficiency months, i.e., months of lessened rainfall. He noted that the drier the weather the greater in general were the number of cases. Seventy-six per cent of the 34 epidemics began in months of deficient rainfall and in 29 per cent of them the dry weather lasted clear through. Twenty-four per cent of the epidemics began in excess precipitation months.

In Cleveland, although September and October of 1926 were the wettest months recorded in 50 years, with a rainfall of 9.10 and 6.75 inches, respectively, as compared to the normal 3.33 and 2.78 inches, the case morbidity was not increased during these months.

TABLE IV

MONTHLY AND ANNUAL PRECIPITATION IN INCHES
1921 TO 1930 INCLUSIVE

YEAR	JAN.	FEB.	MARCH	APRIL	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.	ANNUAL
1921	1.53	2.19	4.39	2.58	1.51	2.38	3.52	3.32	2.77	2.91	3.93	1.75	32.78
1922	1.52	1.31	4.02	2.10	2.42	2.66	3.98	1.20	1.60	1.58	1.37	2.45	26.21
1923	2.61	1.39	1.89	2.21	2.91	1.49	2.09	3.97	3.32	1.73	2.64	4.50	30.75
1924	3.38	1.50	1.65	2.85	2.62	5.73	3.10	0.90	6.08	0.68	0.91	3.58	32.98
1925	1.49	2.42	2.95	1.37	2.24	1.81	4.52	1.42	5.62	3.38	3.60	1.45	32.27
1926	1.78	2.53	1.95	3.44	1.48	2.58	2.86	4.79	9.10	6.75	2.56	2.01	41.83
1927	1.51	2.83	3.62	1.75	3.44	1.63	3.11	0.70	2.74	1.85	7.19	3.91	34.28
1928	2.03	2.36	2.08	2.15	2.04	4.69	6.94	1.15	1.21	3.28	3.25	1.41	32.59
1929	5.23	1.98	3.54	5.26	3.41	4.17	6.30	1.98	1.62	2.68	2.92	2.68	41.77
1930	5.01	2.45	2.68	2.08	1.87	1.84	0.74	1.84	1.69	1.52	2.27	0.89	24.88
Mean Aver. 1871-1930	2.59	2.43	2.76	2.48	3.06	3.26	3.55	2.86	3.26	2.78	2.60	2.39	34.02

When we study the weather data for the past fifty years, we note frequent increases in precipitation as compared to the adopted normals. January of 1874 had 5.16 inches (normal 2.51), August, 1885, 5.14 (normal 2.77), October, 1926, 6.75 (normal 2.78), and December, 1873, 5.50 (normal 2.44). Although the disease was not reportable as such until after 1912, there was no marked increase in deaths due to nervous involvements before that time and one could at least infer that there was no marked increase in this disease during these years. In fact, the precipitation of 1925 and 1930, the years of the greatest local increase in infantile paralysis, was below the normal standard.

Though wetness in itself could not be a factor, there might be some correlation between dryness, high temperature, and the seasonal incidence of the disease.

In 1930, ten months had less than the average precipitation and the monthly temperature figures were normal or slightly above, except in October when it was 1.4° lower than the average of 53.2° F. Some of the months of 1930 were much below the average normal precipitation and the annual precipitation was 24.88 inches, the lowest since 1899 (24.53). High temperature and little precipitation might be factors in increased morbidity, since in 1930 the total precipitation was only 24.88 inches compared to an annual average of 34.02 inches (1871-1930). It

TABLE V

COMPARISON BETWEEN PRECIPITATION AND TEMPERATURE OF THE SPRING MONTHS AND THE MORBIDITY RATE OF POLIOMYELITIS IN CLEVELAND 1912 TO 1930 INCLUSIVE

YEAR	CASES		APRIL MAY	INC.	DEC.	APRIL MAY JUNE	INC.	DEC.
1912	110	Ppt.	623	0.69		819		0.51
		Temp.	1080	39.0		1728	15.0	
1913	34	Ppt.	531		0.23	664		2.06
		Temp.	1052	11.0		1734	21.0	
1914	9	Ppt.	837	2.83		1117	22.47	
		Temp.	1054	13.0		1737	24.0	
1915	143	Ppt.	378		1.76	581		2.89
		Temp.	1060	19.0		1699		14.0
1916	38	Ppt.	447		0.07	645		2.25
		Temp.	1039		2.0	1667		46.0
1917	35	Ppt.	613	0.59		972	1.02	
		Temp.	966		75.0	1614		99.0
1918	33	Ppt.	657	1.03		840		0.30
		Temp.	1102	61.0		1776	63.0	
1919	10	Ppt.	711	1.57		835		0.35
		Temp.	1034		7.0	1768	55.0	
1920	13	Ppt.	613	0.59		1141	2.71	
		Temp.	982		59.0	1656		57.0
1921	36	Ppt.	409		1.45	647		2.23
		Temp.	1137	96.0		1835	122.0	
1922	7	Ppt.	452		1.02	718		1.52
		Temp.	1099	58.0		1785	72.0	
1923	13	Ppt.	512		0.42	661		2.09
		Temp.	1003		38.0	1711		2.0
1924	24	Ppt.	547		0.07	1120	2.50	
		Temp.	977		64.0	1625		88.0
1925	87	Ppt.	361		1.93	542		3.28
		Temp.	1042	1.0		1758	45.0	
1926	25	Ppt.	492		0.62	750		1.20
		Temp.	984		57.0	1627		86.0
1927	69	Ppt.	519		0.35	682		1.88
		Temp.	1044	3.0		1676		37.0
1928	73	Ppt.	419		1.35	888	0.18	
		Temp.	1012		29.0	1652		61.0
1929	32	Ppt.	867	3.13		1284	4.14	
		Temp.	1070	29.0		1728	15.0	
1930	175	Ppt.	395		1.59	579		2.91
		Temp.	1101	60.0		1803	90.0	
Aver-age		Ppt.	554			870		
		Temp.	1041			1713		

is soon seen that such comparisons were fortuitous, inasmuch as in 1925, when the Board of Health case record increased to 87, the precipitation was 32.27 inches and the temperature was 49.7° (average 1871-1930, 49.3°), while in 1927, the year of the next increase, 69 cases occurred and the average precipitation was 34.28 inches, 0.26 inches above normal, and the temperature was 50.8°, 1.5° above normal. The clinching disappointment came when we noted that there were only 7 cases in 1922, a year when the precipitation was the lowest in ten years (26.21 inches) outside of 1930 (24.88 inches) and the temperature was 51.2° (average 49.3°), the third highest in ten years and only 0.6° below that of 1930 with its 175 cases and 1.8° below that of 1921 when only 36 cases were reported.

When we analyze the mean precipitation, the temperature, and the morbidity curves of this disease, we find no relationship when the summer, fall, and winter months are studied as such. We may note, however, what seems to be a relationship when the spring months are grouped. This fact might be suggestive if we should speculate that if weather changes have any effect on the spread of poliomyelitis, such weather changes would have been experienced before the disease made its annual appearance.

To make comparisons easy, we constructed a table in which the following artifices were resorted to. All the precipitation readings were read without a decimal point and added together. The decimal points were likewise dropped from the temperature readings and one lump figure taken. An average was obtained in the same way. The months were divided in 2 groups (Group I—April, May and Group II—April, May, and June). This was done for each year since 1912 and the number of cases noted were those reported by the Cleveland Board of Health. A group that registered below 554 precipitation (actually 5.54 inches) in Group I and 870 (8.7 inches) in Group II was considered as having a low precipitation. Likewise, if the total temperature of Group I fell below 1041° and in Group II below 1713°, the group was considered below normal.

When we examine Table V constructed in this fashion, it would seem at first glance that in years where there was an excess of precipitation in the groupings as we have arranged them, there were no epidemics. Morbidity increases seemed most apt to occur in those years where the precipitation was below normal and the temperature normal or slightly increased. Anything up to 0.5 inches above or below normal was considered within normal limits. The facts could not be correlated well with this table so that another one was made in which the years were grouped in 6 ways (Table VI).

In the first division (A), we placed those years in which an increase in precipitation was shown in both Groups I and II. Such years were

1914 with 9 cases, 1917 with 35 cases, 1920 with 13 cases, and 1929 with 32 cases.

In the second division (B), we placed those years in which the precipitation was normal or nearly so in Group I and above the average in Group II. Such years were actually increased precipitation years and could be grouped with the first division. Such a year was 1924 with 24 cases.

In the third division (C), we placed those years in which there was

TABLE VI

SHOWING THE NUMBER OF POLIOMYELITIS CASES OCCURRING IN CLEVELAND DURING YEARS HAVING INCREASED AND DECREASED PRECIPITATION

TYPE I—INCREASED PRECIPITATION YEARS			
Division A	*Increased Precipitation, Group I*	*Increased Precipitation, Group II*	*No. of Cases*
Year: 1914	+2.83	+2.47	9
1917	+0.59	+1.02	35
1920	+0.59	+2.71	13
1929	+3.13	+4.14	32
Division B	*Normal Precipitation, Group I*	*Increased Precipitation, Group II*	
Year: 1924	−0.07	+2.50	24
Division C	*Increased Precipitation, Group I*	*Normal Precipitation, Group II*	
Year: 1912	+0.69	−0.51	110
1918	+1.03	−0.30	33
1919	+1.57	−0.35	10
		Total	266 (27%)
TYPE II—DECREASED PRECIPITATION YEARS			
Division D	*Decreased Precipitation, Group I*	*Decreased Precipitation, Group II*	*No. of Cases*
Year: 1915	−1.76	−2.89	143
1921	−1.45	−2.23	36
1922	−1.02	−1.52	7
1925	−1.93	−3.28	87
1926	−0.62	−1.20	25
1930	−1.59	−2.91	175
Division E	*Normal Precipitation, Group I*	*Decreased Precipitation, Group II*	
Year: 1913	−0.23	−2.06	34
1916	−0.07	−2.25	38
1923	−0.42	−2.09	13
1927	−0.35	−1.88	69
Division F	*Decreased Precipitation, Group I*	*Normal Precipitation, Group II*	
Year: 1928	−1.35	+0.18	73
		Total	700 (72%)

increased precipitation in Group I with a nearly normal precipitation in Group II. These years could be considered slightly increased precipitation years for the months considered and potentially grouped with the first division. Such years were 1912 with 110 cases, 1918 with 33, and 1919 with 10 cases.

Combining the years with increased precipitation in these divisions, we see that there were 8 years with 266 cases, an average of 38 cases a year.

In the fourth division (D), we placed those years with a definite decrease of precipitation in both Groups I and II. Such years were 1915 with 143 cases, 1921 with 36, 1922 with 7, 1925 with 87, 1926 with 25, and 1930 with 175 cases.

In the fifth division (E) we placed those years in which the precipitation was nearly normal in Group I and definitely decreased in Group II. This decrease in precipitation tends to group these years under decreased precipitation years. The years 1913 with 34, 1916 with 38, 1923 with 13, and 1927 with 69 cases are in this division.

TABLE VII

HOURS OF SUNSHINE IN CLEVELAND

YEAR	JAN.	FEB.	MARCH	APRIL	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.	TOTAL
1921	110	88	140	223	310	333	373	312	221	196	41	58	2405
1922	154	126	156	250	293	327	350	317	283	218	101	103	2678
1923	51	82	177	246	335	351	332	298	202	204	106	79	2463
1924	77	129	134	211	229	292	362	317	188	258	127	46	2370
1925	117	129	202	269	313	332	324	329	200	82	113	73	2483
1926	79	72	159	199	334	337	332	362	159	128	68	42	2271
1927	92	92	149	230	219	313	336	303	256	213	74	70	2347
1928	98	106	197	249	356	215	325	333	270	178	68	105	2500
1929	90	100	189	193	272	331	365	317	225	181	116	59	2438
1930	93	95	191	249	309	330	370	285	250	155	123	61	2510
Average number of sunshine hours as compiled by the local United States Weather Bureau													
	85	103	168	205	265	300	318	282	227	177	92	67	2289
The greatest possible number of sunshine hours as compiled by the local United States Weather Bureau													
	295	297	371	401	452	455	461	429	374	343	295	284	4457

In the sixth division (F), we placed those years in which there was a decrease in precipitation in Group I with a normal precipitation in Group II. These years could be properly classified as decreased precipitation years. The year 1928 with 73 cases belonged to this category.

Grouping the cases of the first 3 divisions of increased precipitation years, we have 266, or 27 per cent of the cases. Grouping the cases of the latter 3 divisions of the decreased precipitation years, we have 700 or 72 per cent of the cases. There were 11 years in the decreased precipitation groups, with 700 cases, a yearly average of 63+ cases. The speculation of decreased morbidity occurring in years of increased precipitation and vice versa would be obvious, were it not for the fact

that in 1912, we experienced an epidemic of 110 cases in a year when the precipitation was increased slightly in Groups I and nearly normal in Group II. It was interesting to note, nevertheless, that most of the cases occurred during decreased precipitation years.

SUNSHINE

When we examine Table VII constructed to show the actual average monthly hours of sunshine and compare this with the ordinary average and the possible hours of sunshine which could be enjoyed under ideal conditions in our locality, we see that most of the months which show a decrease below the average are January, February, and March. The summer months were normal except in 2 instances of the years studied, June of 1924 with eight hours and June of 1928 with 85 hours less sunshine. July and August have always been above the average, while the months of September, October, November, and December showed 17 variations below average. In 1924, the first year that poliomyelitis was increased more than usual in recent years, there were 6 months in which the sunshine was below the normal average as determined by the local U. S. Weather Bureau; while during the epidemic year of 1925, there was a very high average of monthly sunshine, September and October being the only months below normal. Eighty-seven cases were reported during that year. In the next year, 1926, there were eight months in which the sunshine was below par and only 25 cases were reported, while in 1927, with only four months below average, there were 69 cases. In 1929 with four months below average, there were 32 cases, while in 1930 with three months below average, there were 175 cases. Sunshine itself does not seem to be of any significance as regards the morbidity rate.

CONCLUSIONS

1. Ordinary factors associated with seasonal changes, such as temperature, dryness, increased precipitation, sunshine, etc., do not seem to have any effect upon the case morbidity rate of infantile paralysis in Cleveland.

2. There was a decrease in precipitation and a normal or increased temperature curve in all but one of the epidemic years.

3. The morbidity curve shows that the majority of cases of infantile paralysis in and about Cleveland occurred during August, September, and October.

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THE NUTRITIVE VALUE OF STRAINED VEGETABLES IN INFANT FEEDING

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THIS is a report of a clinical study of a series of 60 infants whose diet was supplemented by strained vegetables. Special reference was made to their digestibility and tolerance, and to the contribution of mineral salts and vitamins to the diet.

As Halliburton[1] has pointed out, mere chemical analysis is no criterion of food value, for the digestibility of the food is an equally important question. The results of the test tube experiments are of value but the final test of foodstuffs must be made on animals and preferably on man. There is a growing appreciation of the value of such studies and an increasing resort to their use.

Friedenwald and Ruhrah,[2] state that the digestibility of vegetables depends largely upon the manner in which they are prepared and the amount which is eaten. They also state that green vegetables are valuable, not only on account of the amount of nutriment present in them, but for the large amounts of mineral salts which they contain and their valuable antiscorbutic and other vitamin properties. Vegetables also contain carbohydrates which are well digested and absorbed, and they may therefore be considered as of value as sources of energy, the large proportion of which appears to be available to the body.

Strumpell[3] has shown that in adults about 40 per cent of the contained protein in cooked beans is left unabsorbed when the beans are eaten with the skins, but when the vegetables are finely divided into a flour only 8.2 per cent of the original amount of protein is left unabsorbed. Therefore when vegetables are simply cooked a much larger proportion remains unabsorbed than when finely divided into a powder. The completeness with which a food is utilized according to McLester[4] depends largely upon its physical state, and that proper cooking renders them more digestible.

McCollum has called attention to the fact that in both vegetable and animal tissue the more actively functioning part possesses the vitamins. The leaves which are thin and contain chlorophyl are richest in vitamins. They also contain an abundance of vitamin A, B, C, and E. The leaf is also rich in mineral elements, especially calcium and phosphorus in which elements the diet is often lacking. Spinach is the most

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valuable of the leafy vegetables. It contains vitamin B and C in large amounts, being a valuable source of the latter. It is also rich in mineral elements. The value of peas and beans lies chiefly in the richness of protein. In their dry state they possess pound for pound an even greater amount of protein than meat. As a source of vitamin A they are important. All contain an abundance of vitamin B. They offer a good supply of iron and phosphorus. The carrot is of value because of its high vitamin content. It is relatively rich in vitamin A and also contains an appreciable amount of vitamin C. McCollum further states the value of vegetables lies in their liberal vitamin content and their richness in minerals, which contribute to the formation of the bony structure and teeth. Every element which finds a place in the structure in the cell is essential to life. The applying of this fact to the inorganic mineral components of the food should be emphasized. McCollum continues that it has been estimated that even the abundant supply of calcium obtained from milk is only sufficient to meet the infant's need without providing any natural margin of safety or providing for calcium storage. The growing child must have at least 0.1 gram of calcium a day beyond that needed for maintenance. Under ideal conditions boys are said to consume four times as much calcium per kilo of body weight as is required for men. Fruits and vegetables are relatively rich in calcium. It is estimated that 1.5 grams of phosphorus is the least possible intake which is compatible with safety for the adult. For the growing child who must store phosphorus a much larger amount proportionately, perhaps 2 to 3 times as much is desirable. Clark and Butler[5] point out that a very high percentage of undernourished children show marked evidence of dental decay. They cite the results of examination of 270 children by the U. S. Public Health service, which showed 33 per cent had from 1 to 4 cavities, 48 per cent from 4 to 8 cavities and some had 9, 10, and 11 cavities. McCollum and Simmonds[6] suggest the importance of an effort to change the character of the diet, in order that developmental defects shall grow less common and less severe as time passes. Rypins[7] examined 1197 preschool children in Kansas City, Missouri, between the ages of three and six years. Of these 27.2 per cent had dental caries. Howe[2] has been able to decalcify the teeth of experimental animals and to recalcify them by changing the diet. The decalcifying takes place on diets deficient in vitamin A, B, and C, and adding food rich in these vitamins brings about recalcification.

McKibben[8] states that the reason milk is so good for children is that it is rich in lime. Vegetables and fruits contain mineral salts and vitamins which enable our system to assimilate the lime salts thus laying the groundwork of good bones and teeth. The belief is growing that the decay of teeth is not, as most people believe, caused by bacteria, but by a form of bone degeneration resulting from improper food, and

food lacking in mineral salts and vitamin C, the substances which preserve and build up the bones.

Kohman, Eddy and Gurin[9] state that in a series of studies pertaining to the vitamin content of canned foods, it has been found that each of the vitamins A, B_1, B_2, and C may be supplied in adequate amounts in various canned foods. This has been demonstrated in experiments in which a given canned food was required to be the sole source of one or another of the vitamins. These investigations were carried through five successive generations of experimental animals. The rate of growth and weight at maturity is much higher than that reported as normal. They further state that the rapid rate of growth and maturity weight attained deserve further attention, because either these experimental animals have a more nearly optimum diet for growth and maturity weight than is usual, or there is some heretofore unrecognized advantage in the diet made up wholly of cooked foods as represented by canned foods. They conclude that it is possible that the thoroughly cooked foods are more easily digested and more highly assimilated, making possible a more efficient utilization. The desirability of thoroughly cooked foods for infants is well recognized. The evidence indicates that canned foods may supply every dietary requirement including vitamin B_1 and C so often referred to as heat labile.

Our investigations were conducted in the infant feeding clinic of the out-patient department of the hospital, primarily to discover what foods could be utilized with safety, which would supply adequate minerals in the proper state for utilization as a prophylactic against the future development of mineral deficiency diseases, so admirably described by McCollum. This investigation continued for more than six months, beginning in May, 1931, and ending in January, 1932, and consisted of observation on a series of sixty infants who were fed strained vegetables as a supplement to the regular feeding. The age of these children varied between six weeks and nine months at the time strained vegetables were added to the diet. Twenty-six per cent were under three months of age, 14 per cent between three and five months of age, and the rest over five months of age.

There is a growing appreciation among pediatricians of the desirability of the addition of food rich in mineral content and vitamins to the diet of young infants. Some uncertainty has heretofore been present as to the ability of young infants to digest, absorb, and tolerate supplemental foods of this type. It may be stated that with few exceptions, every child in this series thrived and progressed in a manner which gave evidence that this form of feeding contributed in a large measure to its physical development and well-being. Twenty-seven per cent of the infants were breast fed. The amount of vegetables fed daily was one ounce to infants up to three months of age; from three months to five months two ounces; and five months and older four and

one-half ounces daily. (By weight, two level tablespoonfuls of strained vegetables equals one ounce.) Vegetables were fed in one of two ways, either directly by a spoon or by adding to the formula, since their finely divided state allowed their passage through the nipple.

The stools averaged from 1 to 2 daily, and were smooth, well digested and formed. In only 8 cases were the stools loose, and then only for a duration of two to four days. In one case (Number 21, aged five months, weight 16 pounds, 2 ounces) the mother continued to give strained carrots, 4½ ounces daily, while the stools were loose, and at the end of one week the stools had returned to 1 to 2 formed a day.

There was no vomiting in this series except for two infants who vomited one feeding a day for one month. It was felt that this was not entirely due to the vegetable feeding, as the vomiting stopped although the vegetables were continued.

There was no digestive disturbance or colic other than that just mentioned. There was no restlessness or disturbed sleep.

The vegetables were taken well and liked by the majority of infants. In two cases a few days were required to learn to like the taste of the new food. In only two cases were they refused, and in these two cases it was felt that the mother was not persistent enough.

In order to determine the effect of the iron content of the vegetables upon the hemoglobin and red blood cells, blood counts were made every two to four weeks. In the entire series there was an average improvement in the hemoglobin from 69 to 74 per cent, and the average red cell count improved from 3,757,000 to 3,945,000.

An average gain in weight was maintained by the children in the series, and in some more than the average. Their physical appearance was that of active, well nourished, and developed children with firm muscles and good color.

From these observations it is apparent that vegetables may be fed to the average child, if such feeding is supervised by a competent physician, and that by so doing we are giving infants a food relatively rich in minerals and vitamins which is desirable in order for a growing child to lay the groundwork for good bones and teeth, and supply minerals in adequate amounts for storage. There is no doubt but that in many children there is an inadequate supply of these minerals, since in the home preparation of vegetables the major portion of the minerals and vitamins are often destroyed.

Modern scientific research and investigation enables manufacturers to prepare vegetables in such a manner as to preserve these most important elements. That it is desirable to supply minerals in adequate amounts to infants for the proper formation of teeth and bones is shown in a series of 200 children, selected at random between the ages of two and four years which came under my observation for a period of more than a year. Fifty-two per cent of this series presented decayed, carious

and malformed teeth. In many instances there was lack of proper enamel formation and some teeth were entirely destroyed to the gum margin. It seems that this might be prevented in a large measure, since in private practice where we are able to give the proper and adequate mineral containing foods, the percentage of carious teeth is very small. Infants who are fed strained vegetables receive an adequate supply of minerals for utilization and storage which will provide for the basic needs of the growing child.

CONCLUSIONS

1. Strained vegetables were well tolerated and digested in a series of young infants studied from this standpoint.

2. Digestive disturbances were not encountered, undoubtedly because during the straining process to which these vegetables are subjected, most of the fiber is removed, leaving only the mineral, vitamin, and nutritive elements.

3. The quantity of strained vegetables used was: one ounce daily to infants under three months of age; two ounces daily to infants between three and five months of age; and four and one-half ounces daily to infants over five months of age.

4. Properly prepared vegetables contain an adequate supply of minerals and vitamins to promote optimum development of bones and teeth.

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VON RECKLINGHAUSEN'S DISEASE IN CHILDREN

Report of a Case Presenting Cutaneous Pigmentation and Bone Changes

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VON Recklinghausen's disease is considered to be rather infrequent in children. In recent years, however, there have been several cases reported. In some of these the usual symptom complex of cutaneous pigmentation, nodular formation (neurofibromata), bone changes and mental and physical debility as described originally in the adult type, was present in a modified form.

In the early stages, in childhood, this condition is a monosymptomatic one. The cutaneous pigmentation may be the only manifestation of the disease. This has been termed the "incomplete" form or "forme fruste" of von Recklinghausen's neurofibromatosis. Frequently because there are no associated symptoms, the diagnosis is overlooked. In rare instances, this incomplete form is associated with the typical bone changes of the disease complex. We wish to present such a case and review the literature.

Since 1882 when von Recklinghausen[1] first described this disease numerous reports have appeared with theories as to the etiology, the familial or hereditary tendency, and the relation of the disease to the endocrine system. In 1905 Parkes Weber[2] first presented to the Dermatological Society of London a typical case of what he termed, and which in later years has been more commonly called the incomplete form of von Recklinghausen's disease. However, it was not until 1909 that he fully described this case in a published article. In this description, cutaneous pigmentation was the only manifestation noted. A later report on the same patient revealed tumor formation.

In 1908 Little[3] reported a case in a twelve-year-old boy in whom pigmentation, which was observed at birth, preceded nodular formation by six years. Trimble[4] reported a similar case. In 1919 Adrian and Huegel[5] presented two cases of this form of the disease, one of which developed small nodules in the pigmented areas over the chest and thighs. It was not until after 1921 that cases of this kind appeared more frequently. Comby[6] reported a case in a boy of thirteen in whom there was already tumor formation. Levin[7] also called atten-

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tion to the condition. Acuna and Bazán[8] reported two cases of the disease; one in a girl, aged twelve years, the other in a boy of eleven in both of whom pigmentation was the sole manifestation. Leschke's[9] case was that of an adult female of thirty-two who had pigmentation since the age of five unaccompanied by tumor formation. Ueberschar[10] reported a case in a male of seventeen who had pigmentation at the age of two, and subsequent nodular formation fifteen years later.

Langer and Grumpert[11] have attempted to trace the developmental stages of the disease in four different patients; in one, a twenty-one-year-old girl, there was pigmentation only. Wise and Eller[12] reported two cases, with a paternal hereditary factor, in male children of eleven and nine years of age respectively; the former began to show nodular formation while the latter revealed only pigmentation which was noted in infancy. Goodwin,[13] Acuna and Bazán,[14] Blanco,[15] Jorge and Brian[16] presented cases without any familial history of the disease. Beatty and Cregor[17] reported three cases in female children who had a maternal familial history, with pigmentation only in the older two and nodular formation in the youngest child. The latter had the disease from early childhood while the older children developed the condition in late puberty. Schall, Thibierge, Audry, Hirsch, Feindl, Salomon, Lange, Vignolo-Lutati, and Jadassohn have also reported cases whose initial symptom was cutaneous pigmentation. The accompanying table is a summary of the essential factors of each of the cases reported. The cases with insufficient available data are not included.

Regarding a generally accepted explanation as to the cause of the disease, the literature is entirely lacking. According to Verocay, quoted by Cushing,[18] "the entire process in all probability depends upon an early embryonal disturbance of development of the specific elements of the nervous system which may have affected cells which are capable of producing ganglion, glia, and nerve-fiber cells."

From the summary of the cases reported it may be observed that the disease is as frequent in the male as in the female and that it is first noticed, if not at birth, in early infancy or childhood usually between the ages of two and six.

Preiser[19] has attempted to establish its hereditary tendency; showing "the hereditary factor to be dominant; there being something in the germ plasm that positively facilitates the production, under appropriate stimulation, of tumors of the nerve sheaths." However, in the eight cases described by Acuna and Bazán[14] there was nothing to suggest a hereditary or familial character. In two of the children there was malformation of the spine, in three there was evidence of endocrine derangement, and in two congenital syphilis was plainly manifest. Similarly, in the case here reported, there was no definite etiologic factor, as also will be noted in the vast majority of cases reported in the litera-

TABLE I

AUTHOR	NO. CASES	SEX	AGE	HEREDITARY FAMILIAL FACTOR	AGE PIGMENTATION FIRST NOTED	AGE NODULE FORMATION FIRST NOTED	BONE CHANGES
Weber	1	F	14	none	18 months	14 years	none noted
Little	1	M	12	none	birth	6 years	none noted
Trimble	1	F	19	none	infancy	16 years	none noted
Adrian & Huegel	2	F	14	none	infancy	yes*	none noted
		M	5	none	birth	yes	none noted
Comby	1	M	13	none	infancy	yes	none noted
Acuna and Bazán	2	F	12	none	early childhood	no*	none noted
		M	11	none	early childhood	no	none noted
Leschke	1	F	32	none	5 years	no	none noted
Uberschar	1	M	17	paternal	early childhood	17 years	none noted
Langer and Grumpert	1	F	21	none	early childhood	no	none noted
Acuna and Bazán	8	F	12	none	birth	yes	none
		M	11	none	birth	yes	scoliosis
		F	12	none	infancy	yes	none
		F	7	none	infancy	yes	none
		M	5	none	infancy	yes	none
		M	16	none	early childhood	yes	scoliosis
		M	12	none	early childhood	yes	none
		F	10	none	early childhood	yes	none
Schall	1	M	6	none	infancy	no	none noted
Audry	1	F	15	maternal	early childhood	no	none noted
Hirsch	1	M	14	none	birth	14 years	none noted
Feindl	1	F	18	none	birth	18 years	none noted
Wise and Eller	2	M	11	paternal	3 years	8 years	none noted
		M	9	paternal	infancy	no	none noted
Jorge and Brian	1	F	8	none	early childhood	yes	none noted
Goodwin	1	F	13	none	birth	13 years	none noted
Beatty and Cregor	3	F	14	maternal	puberty	no	none noted
		F	11	maternal	puberty	no	none noted
		F	9	maternal	early childhood	yes	none noted

*Signify nodules present or absent. No available data as to age at their appearance.

ture. Gould[20] has pointed out that since there is occasionally associated some skeletal changes resembling bone softening (osteomalacia) and since this latter condition is considered by many a disease of the endocrines, it may be that these glands hold the secret of von Recklinghausen's disease. Levin[7] and Andueza[21] have also suggested its relationship to the endocrine system.

Clinically, the syndrome is very characteristic. The usual history is that of anorexia, moderate loss of weight, asthenia, and a patchy discoloration of the skin of a brownish (café au lait) color which usually appears early in life. There are, of course, the exceptions. Beatty and Cregor[17] showed two cases in which the disease did not appear until late in puberty. The lesions are flat and irregularly distributed over the trunk, buttocks, and covered portions of the extremities. They vary in size from pinhead to three or four inches in diameter, and are multiforme. They frequently increase in number and in size as the child grows older, but at no time present any subjective symptoms. Darier[22] states that "the pigmentations are of such characteristic appearance especially as regards their distribution on the trunk that when they are the only features present, a diagnosis of this form of the disease is justifiable." Hoekstra[23] is of the same opinion.

Occasionally, there are other lesions associated with this cutaneous feature—namely, the changes in the nerves, bones, and soft tissues. Tumor formation of the cutaneous nerves is the most common and causes few symptoms. Tumors also occur on the cranial, spinal, and sympathetic nerve trunks. The growths may be pinhead in size or much larger, occasionally occupying a whole nerve plexus. When situated on cranial nerves or spinal nerve-roots, they give rise to varied and severe symptoms which suggest morbid growths of the spinal cord or the brain, and the disease may lead to a fatal ending; such cases are, however, extremely rare in children.

The skeletal changes occurring in von Recklinghausen's disease are:

1. Spinal curvatures
   a. Scoliosis
   b. Kyphosis
2. Abnormalities of growth of the long bones
   a. Lengthening—if pathologic condition is in the shaft
   b. Shortening—if the changes occur at the epiphyses
3. Irregularities of the outline of the bone, ranging from central and periosteal cysts to pedunculated subperiosteal tumors.

In the first two groups the lesions are clinically manifest; but in the third type the condition goes unsuspected in the early stages unless detected by routine roentgen-ray studies. Brooks and Lehman[24] published seven cases of the disease in adults in which bone changes were

noted. In two of these there was x-ray evidence, solely, of any bone lesions. These consisted of one small, well-defined clear area in the lower end of each of two femurs and one fibula. It is an identical picture to this type of bone change that the case herein reported presents.

The pathology of this form of von Recklinghausen's disease is chiefly seen in the microscopic section of the pigmented areas of the skin which show a proliferation of the fibroblasts and neurolemma cells which are considered as early neurofibromas. The multiple nerve tumors which

Fig. 1.

appear in the later stages are not connective tissue growths, but, according to Verocay, are composed of a peculiar neurogenous tissue. The nerve fiber cells themselves, or corresponding embryonal cells which possibly have not reached the normal structure of nerve tissue, constitute the basis of the involvement of the bone by the growth of the tumor tissue similar to that in the skin or the nerves. Brooks and Lehman[24] have suggested that with the development of a neurofibroma of a nerve in the periosteum, there is set up a certain amount of reaction and bone destruction, and regeneration follows. If, in the process of the development of the tumor, the osteogenetic element of the periosteum covers

over the tumor, then a thin shell of bone is formed over it and the x-ray appearance of a subperiosteal bone cyst is produced.

Since the disease is more widespread than it was originally thought to be, Parkes Weber's classification must be modified to include the bone changes, not as an anomalous phenomenon but as a definite manifestation of the syndrome.

Fig. 2.

CASE REPORT

P. S., eleven-year-old schoolboy, native American, was admitted to the hospital on October 6, 1931, complaining of a progressive loss of weight, anorexia, asthenia, and a low-grade temperature.

The child's immediate family history was entirely negative as far as any chronic illnesses were concerned. The father and mother were both well, neither having ever had any pigmentations of the skin or any cutaneous nodular formations. The grandparents on both sides were also free of any signs of neurofibromatosis.

He was a full-term, normally delivered infant, weighing eight pounds at birth. At birth it was noticed that the baby had several (about a dozen) brownish pigmented areas over the trunk. His development was normal. At one and one-half years of age he had mumps and a year later had measles. Between three and five years of age he had frequent attacks of "sore-throat with high fever." These did not recur after a tonsillectomy was performed in his fifth year.

It was at this time that the parents noticed that the child had been getting many more small brownish pigmented areas, scattered discreetly over the entire body in rapidly increasing numbers, while some of the old spots were getting larger. There

Fig. 3.

were no other symptoms present. These cutaneous lesions slowly progressed. It was not until he was eight and one-half years old that his appetite began to wane and he commenced to lose weight. The anorexia and loss of weight continued steadily to the time of admission when he weighed fifty-two and one-half pounds, having lost approximately twelve pounds in the two and one-half year period. His mental development was normal.

Physical examination revealed a white male child of the linear type, very poorly nourished, who, though he did not complain nor appear uncomfortable, looked as though he were suffering from some chronic illness. He was extremely alert, reacted quickly, and cooperated most understandingly. His mentality was above the average for his age. The color of his skin was somewhat dusky. His head appeared large; the suboccipitobregmatic circumference was 47.5 cm., while the oc-

cipitofrontal was 49. cm. The scalp was normal as were the eyes, ears, and nose. Examination of the fundus oculi was also negative. The throat revealed a mucopurulent discharge in the postnasal area. The neck offered a few palpable paratonsillar lymph nodes bilaterally, but there were no masses or abnormal pulsations. No thyroid gland was made out. In the chest the lungs were clear throughout. The heart presented a systolic murmur at the apex, not transmitted nor affected by posture or exercise. The sounds were of good quality; the second pulmonic was accentuated. There was no apparent enlargement of the heart. The abdomen was moderately sunken and there were no masses or abnormal pulsations palpable. The liver was not felt but the spleen was made out 3 cm. below the left costal margin. After the tenth day in the hospital it was no longer felt. The extremities were apparently negative and the reflexes normally active. The genitalia were normal. There were a few small lymph nodes palpable in both inguinal regions. Discreetly scattered over the neck, axilla, chest, abdomen, back, and buttocks were numerous macular light brown (café au lait) pigmentations of sizes varying from pinhead to 7 cm. in diameter and irregular in shape. There was no evidence of the slightest cutaneous nodular formation anywhere on the body. Figs. 1 and 2.

LABORATORY DATA

The Schick test was negative. The von Pirquet reaction was repeatedly negative as was also the Mantoux test. The blood pressure readings averaged 116/58. Urine examinations were always normal and the blood Wassermann was negative. The blood picture, an average of the many counts done, was as follows:

Hemoglobin		70%
Red Blood Cells		2,900,000
White Blood Cells		8,200
Polymorphonuclear Neutrophiles		51%
	Basophiles	2%
	Eosinophiles	5%
Lymphocytes		22%
Mononuclears		20%
Blood Platelets		120,000
Vital Stain Smear		no reticulocytes no abnormal cells
Fragility Test		0.38% to 0.44%
Coagulation Time		5 min.
Bleeding Time		2 min.
Icteric Index		4
Van den Bergh		negative

Blood Chemistry:	Urea nitrogen	14.0 mg. per 100 c.c.
	Creatinine	1.0 mg. per 100 c.c.
	Sugar	80.0 mg. per 100 c.c.
	Cholesterol	200.0 mg. per 100 c.c.
Basal Metabolism:	Plus 23%	

Roentgen-ray examination of the chest, skull, and genitourinary tract were entirely normal except for the right maxillary sinus which was slightly cloudy. The left femur showed a small, oval area of lesser density on the medial aspect of the distal end (Fig. 3). On repeated examination the same lesion was noted.

At the time that his antrum was cloudy he ran a low-grade temperature for four days, at no time exceeding 100.2° F. This returned to normal with a corresponding subsidence of the sinus condition. Exactly one month after admission he was discharged, to be followed up in the out-patient department. His general condition had markedly improved. He had gained five pounds in weight and his appetite remained excellent. Examination was entirely negative except for the cutaneous pigmentations.

COMMENT

Before establishing a diagnosis of the incomplete form of von Recklinghausen's neurofibromatosis in this case, it was necessary to rule out definitely two conditions whose skin manifestations are occasionally confusing. Urticaria pigmentosa was excluded by the absence of the elevated cutaneous lesions, wheal formations, and pruritus. Also, the onset of the disease and the characteristic distribution of the lesions were unlike that seen in urticaria pigmentosa.

In this instance, differentiation is made from Addison's disease, because of the lack of generalized bronzing and pigmentation of the mucous membranes, persistently low blood tension, gastrointestinal symptoms, and marked general debility.

In classifying our case, we feel that it would be improper to include it in the "incomplete" group, where the only manifestation is skin pigmentation, inasmuch as there were bone changes present. It is also very probable that bone lesions would have been observed more frequently in the cases cited above had complete roentgen-ray studies been made in those cases which presented no clinical evidence of skeletal changes.

No conclusions can be drawn substantiating the prevalent opinion that this is an hereditary familial disease. In the cases summarized, there were only four instances with a familial background. It is interesting to note, however, that in each instance the sex of the patient afflicted was the same as the parent who had evidence of the disease.

In the majority of the cases, the mentality of the patient is not affected unless there is some distinct endocrine derangement present.

SUMMARY

1. A case of von Recklinghausen's disease characterized by cutaneous pigmentation and bone changes without skin tumors is presented.

2. In the review of the literature, the hereditary factor of the disease cannot be established.

3. Mental derangement is not a common symptom of this type of von Recklinghausen's disease.

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ANTIQUES OF PEDIATRIC INTEREST

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THE pap warmer consists of a perforated base, supporting a covered double boiler. The heating element was a small alcohol lamp. This apparatus was used to keep the pap warm for feedings which were usually so frequent that they brought about the pukings which if they were absent was thought by many to be a sign that the infant was about to go into a decline.

Pap was essentially a thick feeding, prepared by mixing or cooking flour or bread with water. Occasionally milk was added, but some

Pap warmers, circa 1830

Left, Wedgewood pottery; Painted tin, American; Transfer printed, Staffordshire pottery, English. Height 10 inches.

authorities considered this addition dangerous. It might bring on the watery gripes, or the infant might imbide with the milk the evil passions and frisky habits of the animal supplying the milk. As a substitute for milk or water pap, Brouzet, 1754, recommended the use of pap as prepared by Van Helmont. This was made from bread lightly boiled in beer or wine with added meal or sugar.

Detailed directions for the making of pap are given by Raulin, 1769.

"One makes good pap with light bread well baked and well fermented. The leaven divides and reduces the tenacious particles of the

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flour. The fermentation which it produces in the paste disengages a large part of the air which was held there. It is this that renders pap so made more proper than all others for nourishing infants. One prepares it by boiling bread broken into morsels in water into which one puts a little oil or butter following the usage of the country. One reduces it over a gentle fire to the consistence of a jelly or of a light panada. The bread ought not to be freshly baked; it is better when three or four days old. One ought to smooth the pap with brown sugar or honey or indeed with loaf sugar if the infants are threatened with diarrhea. One also makes pap with wheat biscuit which one knows under the name of sea biscuit. One cooks it with pure water in an earthen vessel stirring with a wooden spoon until the material is reduced to a jelly. In place of biscuit one slices well-fermented bread, and dries these slices at the fire. One powders it and keeps it in bottles or other vessels in a dry place. When one wishes to serve this powder one takes an amount of powder suitable to the quantity of pap which one wishes to prepare, one softens it with lukewarm water and reduces it over a low fire to the consistence of jelly. One regards with reason the flour of malted wheat as very good to make pap. It is prepared like the preceding.

"One ought to fear that pap made by the admixture of milk would become more likely to cause colics than it is by itself."

Critical Review

DISORDERS OF INTERNAL GLAND SECRETION IN CHILDREN

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ALTHOUGH thousands of papers are written on the glands of internal secretion, as can be seen in a casual survey of the Quarterly Cumulative Index, surprisingly little can be accepted as established fact. The most important of the facts that can be accepted are as follows:

The following hormones have been identified, isolated, and their actions determined. Pituitrin (1895) is derived from the posterior lobe of the hypophysis; it is concerned with the fat metabolism, water metabolism, heat regulation, and possibly sleep. Adrenalin (1901) is derived from the medulla of the suprarenal glands. It produces a variety of effects which are the same as those resulting from stimulation of the sympathetic nervous system. Thyroxin (Kendall, 1917), the active principle of the thyroid gland, affects both growth and the heat production or energy metabolism. It accelerates calcium and phosphorus metabolism, and has an influence on the growth of all systems of the body. Its action is intimately connected with iodine, and it is by the iodine content that the therapeutic strength of a preparation of thyroid used is graded. Although the pure principle thyroxin is only of recent origin, extracts of the thyroid gland have been used beneficially during the last forty years. Insulin (Banting and Best, 1921) is derived from the islands of Langerhans of the pancreas, and is necessary in the combustion of sugar.

Active substances have also been separated from the ovaries, parathyroid glands, anterior lobe of the pituitary, and adrenal cortex. The following names are rapidly becoming familiar in the literature—parathormone (Hanson, 1923, and Collip, 1925) from the parathyroids; the growth hormone of the anterior lobe of the pituitary; the sex hormone from the same origin; follieulin, pitocin and pitressin fractions of pituitrin, progestin and theelin from the ovary; emmenin from the placenta; and cortin from the suprarenal cortex (Swingle, Pfiffner, and Hartman).

There is great confusion in the clinical literature relating to abnormalities of the glands of internal secretion because of the loose use of terms and the way they are combined. It is not surprising that this has happened, because the physiologic laboratories have given much proof of the intimate interaction of various glands. This knowledge has not been developed sufficiently for accurate application in clinical practice and the clinician should be satisfied with diagnoses of single glands. Added difficulties come from improper diagnoses, examples of which are not uncommon. Photographs of typical Mongols are given as examples of thyroid, or combined deficiencies in at least two authorita-

tive systems of endocrinology. Studies of the basal metabolism of Mongols have shown that the thyroid is not involved in the majority of cases, and then only to a minor degree. This makes one wonder how much dependence can be placed upon the other diagnoses of combined glandular involvement which are filling the literature. The safest method to approach the problem is to consider each gland individually and to plan the treatment of a case according to the gland which is primarily involved.

Growth is markedly influenced by various endocrine glands. Since it is the essential feature which separates childhood from adult life, it is of especial importance in the recognition and diagnosis of endocrine disturbances of childhood, and in the adult the result of earlier abnormal growth is significant. Overaction of the pituitary, pineal, sex glands, and adrenal cortex results in superior or excessive growth, while underaction of the thyroid, gonads, and pituitary results in deficient growth or dwarfism. The effect on growth of various parts of the body differs with different glands. In hypothyroidism, for example, the delayed growth is seen most in the extremities, while at least one type of eunuchism may be characterized by overgrowth of the extremities. The latter is an exception to the general rule.

In order to be able to recognize whether growth of the body as a whole or of a part has departed beyond normal limits, standards of comparison are necessary. These can be found either in articles by the writer[2] or Engelbach,[3] and a study of deviations from normal growth is an important as any laboratory test. This is recognized by Engelbach who gives it an important place in his recent book.

LABORATORY TESTS

1. *Basal Metabolism.* Determination of the basal metabolism is essential in the diagnosis and treatment of disease of the thyroid gland. It is pretty nearly diagnostic during childhood, but the fact that hypogonadism in the female, and the rare case of deficient secretion of adrenalin may also result in a low metabolism, must be borne in mind.

The writer stated in his book on *Internal Secretion and Metabolism*[4] that the basal metabolism of obesity seen in deficiency of the anterior pituitary was slightly minus to minus 20 per cent. A recent unpublished study of these cases shows that these conclusions were incorrect, and that the metabolism is normal and not subnormal in the majority of instances. This error was due to the fact that dependence was placed on the formulas used in estimating the surface area of the body. A recent unpublished study has shown that the body surface derived by the height-weight formula, when compared to that obtained by the DuBois linear formula, may have maximum deviation of minus 26 per cent, and an average deviation of minus 8 per cent. Errors may appear in this way in cases with abnormal deposits of fat or edema, or in cases of severe malnutrition with complete loss of body fat or dehydration.[4]

2. *Pituitrin and Adrenalin Reactions.* Engelbach[3] concluded from his experience with a large series of cases that the variability of these reactions was so great that no practical deductions could be drawn from them.

3. *Blood Chemistry. Sugar.* It is unnecessary to discuss the part played by the circulating blood sugar in diabetes mellitus. The sugar

tolerance curve, as well as the threshold, are of diagnostic as well as therapeutic importance. The sugar tolerance is lowered in diabetes mellitus, and is increased by insulin. There is considerable doubt as to whether the technic of determining the sugar tolerance has been sufficiently studied in normal individuals to make the test useful as a diagnostic procedure in other endocrinopathies. This is particularly true of childhood in which the dosage of glucose must vary according to the weight. It explains the great divergence of results reported. The final word has not yet been written which decides whether pituitary or gonadal obesity is due to a very high sugar tolerance or not.

4. *Calcium.* Although the blood calcium tends to be lowered in congenital cretins, this finding is of no diagnostic significance. The calcium metabolism is intimately related to the parathyroid glands, which have a specific effect on the calcium of the blood. The injection of parathormone results in an increase in the concentration of calcium circulating in the blood. Parathormone cannot be used indiscriminately without testing its effect on the blood, because in animals the calcium has been forced up to such a height that death results. In such cases the blood may become so thick that it can no longer circulate efficiently. The possibility that overdoses might also result in death to the human being has not been ruled out. A deficiency of calcium accompanies tetany, due either to lack of parathormone, or to lack of vitamin D as in infantile tetany. The former is rare in childhood, while the latter is common. Although low blood calcium is almost diagnostic, especially when there is a concurrent lowering of the blood phosphorus, final evidence can be obtained in some cases only by determining the calcium balance (Bauer and Aub).[5]

The blood nonprotein nitrogen and the test for phenolsulphonephthalein excretion tests have according to Engelbach[3] only a relative and insignificant value.

Thyroid Gland.—Abnormal function of the thyroid gland appears either as overactivity or deficient activity. When the gland is overactive it increases in size. It may be enlarged, however, without being overactive. This is known as colloid goiter.

Colloid Goiter.—Such enlargements appear in all parts of the world where the supply of iodine is low and they are intimately connected with the iodine intake. They may be prevented, as shown by Marine, by giving two grams of sodium iodide each spring and fall in doses of 30 centigrams twice daily. Colloid goiter is especially common during adolescence and appears even in the presence of sufficient iodine. Means[5] explains this as probably due to "some error in assimilation." Further investigations are necessary to answer this question, as the enlargement may be connected in some way with the profound changes taking place in the gonads at puberty. Usually this type of thyroid enlargement requires no treatment beyond making up any iodine deficit.

Exophthalmic Goiter is very rare in early childhood, and the youngest recorded case is three and one-half years old. It responds so well to medical treatment that surgical interference is rarely necessary during childhood. In this respect it differs from adult toxic goiter. Naturally, the longer a goiter persists and the older a child is, the more indication there is for surgical interference. It is better judgment to postpone such treatment until after puberty whenever possible. The thymus is

frequently enlarged in these patients and treatment of it by x-ray has been of some help in controlling the symptoms of hyperthyroidism.

Another rare condition known as *Adenomatous Goiter* does not vary in its characteristics from that seen in the adult. The surface is nodular and iodine is contraindicated.

Enlargement of Thyroid With Hypothyroidism.—Enlargement of the thyroid gland may be accompanied by a low basal metabolism in those instances where there is destruction of the functioning tissues of the gland, as for example, cysts. This condition is rare in children in this country. Its treatment depends upon the size of the gland and the degree of hypothyroidism. Both surgery and medicine may be indicated.

Hypothyroidism.—This condition is characterized by a deficiency of thyroxin, lowered basal metabolism, and retardation of growth. The need of an early diagnosis and treatment has been emphasized by the writer. Since it is reasonable to believe that the longer treatment is delayed the less opportunity there is of attaining complete normality. The disease is recognizable in the third month of life. Results of treatment have been more encouraging from the physical than the mental point of view. The different preparations of thyroid extract on the market vary in strength, and it is important that the general practitioner knows which form he is using. Thyroxin is the most potent and is between 2000 and 3000 times stronger than the weakest of the preparations of thyroid.

Myxedema appears later in childhood. It responds more satisfactorily to treatment than does cretinism, because some years of normal growth have taken place before there is a deficiency of thyroid. The dosage of thyroid for each individual depends upon the severity of the condition. The basal metabolism can be measured to determine its effect.

Puberty.—There is an increase in the basal metabolism at puberty which can be considered physiologic. It is found in both sexes[6] and may reach plus 35 per cent. This increase is apparently temporary and disappears after puberty is established; it may be analogous to that found in adults during the menstrual period. Topper[7] also made a study of the increase in metabolism sometimes found in children between 6 and 14 years with simple goiter, and concluded that this also disappeared when puberty is established. Warning is given not to diagnose such cases as exophthalmic goiter and treat them unnecessarily.

Another type (unpublished data) was found in girls from fourteen to eighteen years of age. These girls have a distinctly lowered metabolism which on the average is minus 20 per cent and are without any of the classical signs of hypothyroidism. Although it is not yet clear whether this group represents true hypothyroidism or is due to lack of iodine, or some physiologic change, it has been found that the general health is benefited by the administration of thyroid. This group will be reported and discussed in a separate paper.

Thymus.—Although the thymus is known as a gland, no one has been able to prove that it has an internal secretion. Its function is obscure and experiments on animals have demonstrated that it is not necessary for life. Its absence does not influence growth.

Enlargement of the Thymus.—Although the pathologist may set an arbitrary weight above which the thymus is believed to be enlarged, there is no convincing data which fixes the normal limits of weight of

this organ. Further confusion has arisen from x-ray pictures which show only one diameter of the organ. Errors have arisen from failure to take into consideration how the shape and shadow of the thymus changes with respiration. Little or nothing is known as to what extent its size and shape may change during life, due to engorgement with blood. It must be concluded, therefore, nothing is known about the thymus as a gland of internal secretion, and that probably most of what was thought to be known about its relation to such conditions as thymic asthma and thymic death is unsubstantiated and without scientific proof.

Pineal Gland.—The reviews[8, 9] of the literature show that very little has been added to the knowledge of this subject. Pineal tumors have been associated with precocious development of the body and the genitals. X-ray examination is generally of little help and the diagnosis is usually made at autopsy. Symptoms of pineal tumor usually do not manifest themselves until the tumor is large enough to cause symptoms of intracranial pressure. Because of this and the rarity of the disease during childhood, the knowledge of the condition, particularly in its early stages, is very scanty.

Adrenal Gland.—Each adrenal gland consists of two parts, the medulla and the cortex, which differ in their embryonic origin, functions, and chemical composition.[5] The gland is very vascular and may vary in size under different conditions. In some cases, both in children and adults with marked accentuation of the male characteristics, the cortex is relatively large, while it is deficient in Addison's disease.

The secretion of the medulla, known as adrenalin, is thoroughly understood. Cannon[10] has shown in a brilliant series of investigations its close relation to the sympathetic nervous system. The hormone adrenin produces a variety of effects which are identical with sympathetic stimulation when it is introduced into the blood stream. "The pupils are dilated, the glands having sympathetic innervation are stimulated, the bronchi are relaxed, the heart is stimulated and the gastrointestinal tract is depressed. The blood vessels of the skin and most mucous membranes are constricted and those of the skeletal muscles are dilated. The net result of all these effects is to integrate the body for strenuous activity, causing increased blood pressure, increased discharge of carbohydrate into the blood stream, and a differential augmentation of the circulation of the nervous system, heart, and skeletal muscles. These facts form a basis for Cannon's emergency theory of adrenal medulla function."[5]

The secretion of the cortex, known as cortin,[1] is as yet too new to be completely studied and understood. It has already given as brilliant results in the treatment of Addison's disease as insulin has in the treatment of diabetes mellitus.

Tumor of the adrenal cortex, like that of the pineal gland, is associated with precocious mental, physical and sexual development. In the female, it is also said to evoke the male secondary sexual characteristics.

Since Addison's disease is almost unknown in children, and the knowledge of this condition is almost nil, it is possible that deficiency of the adrenal cortex may manifest itself in the early ages by a different group of symptoms and signs from those seen in the adult. It would be a brilliant addition to the therapeutic armament if definite indications could be discovered for the use of cortin in pediatrics.

Adrenin has uses which are well known and therefore will not be discussed in this review.

Tumors.—Tumors of the adrenal glands are more common in the female than in the male. Adrenal carcinoma and sarcoma are usually seen in children less than four years of age. Their existence is not usually suspected until metastasis through the lymphatics causes symptoms which appear quite unrelated to the primary lesion. It is always fatal. The fact that they practically never produce symptoms which can be attributed to functional abnormalities of the adrenals may be explained by their rapid development and the short course of the disease.

Tumors of the suprarenal cortex may be either malignant or benign, and may produce the condition which has given rise to the term "infant Hercules." It is never seen in girls.

Pituitary Gland.—Disturbances of the pituitary gland and the neighboring tissues have led to many investigations which have added much to our knowledge, but have not been sufficiently clear-cut to definitely separate the different parts of this organ. It has been found convenient, however, to speak of the anterior and posterior lobes because it is possible to derive independent extracts from these two parts. Injury to the posterior lobe indicates that it has something to do with "the fat metabolism, the water metabolism, heat regulation, and possibly sleep"[5] while the anterior lobe is concerned with growth and sexual activity. The two lobes are so closely confined in the sella turcica that injury to one without the other is unlikely.

Pituitrin is derived from the posterior lobe. There is sufficient evidence to connect lack of pituitrin with the polyuria of diabetes insipidus. The extract of the posterior lobe can be separated into a portion which influences the blood pressure and another fraction which acts on the smooth muscles of the uterus. These are known respectively as surgical pituitrin and obstetric pituitrin, and have been designated venopressin and oxytocin. The injections of pituitrin are also reported to lower the blood lipoids and cause a deposit of fat in the liver.[5] Injections of pituitrin in the ventricle are more effective than when introduced into the blood stream, but the difficulties of so doing are obvious.

Complete extirpation of the pituitary gland usually results in "failure of growth, adiposity, genital infantilism, defective development of the adrenal cortex and thyroid, and often hypothermia and polyuria."[5]

Injections of the growth fraction in animals results in true gigantism until closure of the epiphyseal junctures prevent further growth, after which acromegaly develops. The former is seen during childhood, while the latter develops in adults.

It is obvious that many combinations of abnormalities of the pituitary gland are possible which explain the presence of numerous descriptive diagnoses and combinations of diagnoses in the literature. This has led to much confusion which will not be cleared up until the physiologists and pathologists give us a firmer basis on which to build.

Gigantism is believed to be abnormal growth in height, originating in childhood, due to excessive secretion of the growth hormone of the pituitary. Acromegaly is due to excess of the same hormone after the epiphyses are closed. A description of both types may be found in textbooks. Dickens recognized the late stage of muscular weakness in the *Old Curiosity Shop* where he says, "Giants are of no use in the

circus after they have become weak." These two conditions, however, are more important in adult life than during childhood.

Dwarfism.—Little is really known about dwarfism, although its etiology is possibly a hypoplasia of the anterior lobe of the pituitary gland. Much has been written but as yet most of the work is either descriptive or in the realm of speculation. The failure in growth seen in cretins should not be confused with that seen in dwarfs without hypothyroidism.

Dystrophia Adipose-Genitalis.—The famous Fat Boy of Dickens' is a classical example. The condition is not rare and may appear suddenly during the first year of life, as it did in one of the writer's cases. It is due either to a disturbance of the hypothalmic region of the brain or the hypophysis, or both. The subjects are obese, have undeveloped genitals, and the excessive fat may have a female distribution in the male. The breasts are often markedly enlarged. There seem to be two types, one with exceptional minds, and the other with backward mental development. The former responds satisfactorily to treatment. The condition may progress into adult life or it may disappear spontaneously. Contrary to the current belief, the basal metabolism of children with this condition is rarely, if ever, below normal and in consequence one can conclude that there rarely, if ever, is involvement of the thyroid gland (unpublished data of writer).

The difference between simple nonglandular obesity and dystrophia adipose-genitalis is not clear-cut and it is often impossible for a clinician to definitely place a case in one class or the other. If the symptoms include any abnormality in heat regulation, the water metabolism (diabetes insipidus), growth, sexual development, or activity, the case is probably due to a deficiency of the pituitary. The x-ray rarely helps in diagnosis. The physical signs of tapering fingers and a female distribution of fat in the male may be of some significance, but they cannot always be depended upon.

Diabetes Insipidus may be a symptom of any type of pituitary involvement. It is characterized by marked polyuria and polydipsia. The urine is of a very low specific gravity, and the kidneys are unable to concentrate it. The health and urine are otherwise normal. It is possible to reduce the urine output from as high as ten liters to one liter a day by the subcutaneous injection of pituitrin.

Various other types of pituitary involvement are so rare that they will not be discussed.

Gonads.—There is a sex differentiation in the amount of heat produced by the two sexes which reaches its maximum at puberty. This with other obvious differences in physique suggests that the hormones of the gonads begin to differentiate and to be active at an early age, possibly at the end of the first year.

Testes.—The effect of castration becomes evident at puberty. The epiphyseal lines remain open and the long bones continue to grow—even into the third decade. This results in abnormal growth of the arms and the legs to such a degree that their disproportion may be of diagnostic importance. The voice remains high-pitched; some subjects remain thin, while others become obese. The situation is often accompanied by changes in character.

A testicular material has been obtained, the active fraction of which is in the lipoid substance. It is effective both by mouth and injection.

Under some circumstances testicular grafts have lived and have compensated in part for the deficiency of the testicular hormone.

Precocious Puberty has been attributed to pineal tumor, adrenal tumor, and the gonads. It is little understood.

Ovary.—At puberty two definite hormones are definitely recognized (a) the follicular hormone, and (b) that of the corpus luteum.

Eunuchism may be present in the female, as well as the male, and presumably has the same overgrowth of the extremities and other general characteristics. In the matured female, functional hypogonadism is accompanied by a minus 15 per cent reduction in the basal metabolism, a slight reduction in body temperature, and a moderate reduction in blood pressure. With this there is a 50 per cent reduction in the galactose tolerance.[11]

Allen and Doisy[12] found that injection of follicular fluid had a profound influence on sexual processes, and the latter was able to isolate what seems to be a true hormone. This is relatively ineffective by mouth.

Allen[13] isolated a true hormone of the corpus luteum "progestin." It is antagonistic to the follicular hormone, although both have a stimulating action on the uterus and mammary glands.

Menstruation.—There may be a specific rise in the metabolism connected with the menstrual period. This should not be confused with pathologic conditions.

Precocious Puberty.—Sexual precocity commences most commonly in the second year, but may appear at birth. The external genitalia may enlarge rapidly, and the growth and distribution of hair resembles that of the adult. Intelligence is nearer the actual than the sexual age.

Interrelations of Endocrine Organs.—Although there is considerable proof of such interrelation, the clinical data is so conflicting that it is not yet possible to correlate the various syndromes on a proved scientific basis. It is, therefore, more rational to approach a problem from the point of view of individual glands, and not to confuse the picture with double or multiple diagnoses. It is only in this way that this fascinating realm of medicine will be removed from the Wonderland of Alice, and placed on the sound basis demanded by scientific medicine.

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SECOND ANNUAL MEETING OF THE AMERICAN ACADEMY OF PEDIATRICS

FRIDAY AFTERNOON SESSION
May 13, 1932

Round Table Conference on Scarlet Fever

A Practical Consideration of Skin Tests, Active and Passive Immunization and the Use of Therapeutic Antitoxins

Leader: John A. Toomey, M.D.; Assistants: Archibald Hoyne, M.D., and A. Clement Silverman, M.D.

In this seminar, we are asked to consider the subject of scarlet fever. To cover this subject completely would take more time than we have at our disposal. Therefore, we have divided it into three topics as follows:

I. Skin Tests in Scarlet Fever (Introduced by Dr. Silverman)—
   A. The Dick Test
   B. Schultz Charlton Reaction
   C. Conclusions

II. Immunization (Introduced by Dr. Toomey)—
   A. Active Immunization
   B. Passive Immunization
   C. Conclusions

III. Treatment (Introduced by Dr. Hoyne)—
   A. The Therapeutic Use of Scarlet Fever Antitoxins
   B. Conclusions

I. Skin Tests in Scarlet Fever

A. The Dick Test.—For clarity, one need only mention that the Dick test is a test to determine the susceptibility of an individual to scarlet fever and that its discovery by the Dicks has been of exceeding importance to medicine.

Most individuals who use this test routinely have some doubts as to its absolute specificity since many people develop scarlet fever who have had negative Dick tests. There are likewise people who have had scarlet fever who still have positive Dick tests at the end of their illness 30 days later.

It might be said of the former cases that such individuals have had a natural loss of immunity, as a result of which they had become susceptible to scarlet fever. That this cannot always be the case is seen from the fact that these patients still have negative Dick tests at the beginning of the scarlet fever rash when, theoretically, they should have positive tests. The actual percentage of these negative Dick reactors who develop scarlet fever is not known, but our experience indicates that such cases are not rare.

There is another group of scarlet fever patients who never have had Dick tests previous to admission to the hospital, but who show negative Dick reactions on admission at the height of their rash and when theoretically they should have a positive Dick reaction. There are so many of these cases admitted to our hospitals that the finding of a negative Dick test at the height of a rash cannot be said to rule out scarlet fever. It is very probable that the rash in these instances is caused by other strains than those originally described as being the sole causes of the disease.

Certainly, the strain that causes scarlet fever one year may not be that which causes an epidemic of the disease the succeeding year since the majority of patients of the first year may be Dick positive and the majority of the second, Dick negative.

DR. E. B. SHAW, SAN FRANCISCO, CALIFORNIA.—I agree with the conclusion of Dr. Silverman and wish to state that in my experience there was a definite lack of correlation between the blanching findings and the Dick test itself. I should like to ask whether the Dick test can be definitely stated to be a toxin reaction? The response of the skin to a Dick test is hardly the same as a Schick reaction. It begins promptly and gradually reaches a maximum in twenty-four hours. It is not characterized by the latent period so characteristic of the tissue response to a true toxin. Furthermore, there has not, to my knowledge, been shown the same degree of consistency between the Dick reaction and the immune state to scarlet fever that one encounters with regard to the Schick reaction and immunity to diphtheria. Is not the Dick reaction somewhat intermediate in its nature between a toxin and an allergic skin response?

DR. J. S. WALL, WASHINGTON, D. C.—Perhaps the percentage of errors that occur in interpretation of the Dick test may be due to the time of reading the test. I suggest that waiting a day or so more might reveal a number of delayed reactors. I feel that it might be very difficult to interpret Dick tests at the height of the rash.

DR. A. C. SILVERMAN, SYRACUSE, NEW YORK.—The Schick test differs from the Dick test in that the reaction persists longer. The former material is less stable than the latter.

DR. C. A. EARLE, DES PLAINES, ILLINOIS.—I have given over 11,000 Dick tests during the course of my work in several institutions. I have seen only one negative Dick reactor develop scarlet fever. All those who have had scarlet fever, in our experience, have always had a negative Dick test. I have great confidence in the Dick test as evidence of susceptibility and immunity to this disease. There may be some physical reason to explain the number of negative Dick tests that occur in many people who should be positive according to the history.

DR. J. A. TOOMEY, CLEVELAND, OHIO.—I think that the Dicks have shown that many minor apparently innocuous physical things may cause destruction of the toxin. When one remembers that 1 c.c. of 4 day toxin may contain as high as 6,000 or more skin test units, one can understand how easily physical reactions might destroy this minute amount of toxin. Too much alcohol, tincture of iodine, antiseptics of various kinds can vitiate the test. Great pains should be followed in the care of needles and syringes. Both should be dry sterilized, since even the small amount of moisture and water of condensation that collects after cooling might alter the test reading. Paying attention to all these points, we still have negative Dick test reactors in individuals who should be positive. We feel that the belief that there are only two strains of specific streptococci that cause the disease entity of scarlet fever is entirely erroneous.

B. Schultz Charlton Reaction.—These authors discovered that convalescent scarlet fever serum injected into the skin of an acutely ill scarlet fever patient blanched

the rash about the injection. They concluded that a specific product had appeared in the serum of convalescent patients which brought about a local cure of the disease and that the reaction was specific.

It might be interesting to mention that the specificity and the worth of any antitoxin is often gauged by its ability to locally blanch the rash of an acutely ill scarlet fever patient. If this criterion is true, there are many antitoxins now on the market that are worthless since many merely cause a local inflammatory reaction and no blanching whatever.

One cannot say that a rash is not a scarlet fever rash just because scarlet fever antitoxin does not locally blanch this rash, for we have had hundreds of cases in which the rash has not been blanched by scarlet fever antitoxin and has been by coincidentally injected convalescent scarlet fever serum. These patients have had positive Schultz Charlton tests with convalescent serum, a negative reaction with scarlet fever antitoxins and, curiously enough, many have had a negative Dick test at the same time. We feel that we cannot be too specific about conclusions in regard to the Schultz Charlton phenomenon and although a positive Schultz Charlton phenomenon obtained with convalescent scarlet fever serum is fairly conclusive, yet its absence does not conclusively rule out the disease.

C. Conclusions.—We feel that the Dick test is a very valuable aid in detecting susceptibility to scarlet fever, but we do not think that it is an absolutely perfect test. We feel that a negative reaction does not rule out the possibility of scarlet fever in individuals with scarlatinal rashes. We would say, however, that the exceptions are not innumerable and that for practical purposes, one could ignore them, merely keeping in mind the limitations.

A positive Schultz Charlton reaction is good evidence for the presence of scarlet fever, but a negative Schultz Charlton does not rule out the disease.

DR. WALL.—The Schultz Charlton tests can often be seen and appreciated better if read at a distance rather than closely.

DR. SHAW.—The Schultz Charlton reaction is of no great value. It is no good in doubtful cases and certainly not needed in typical ones. I agree with the speaker when he states that convalescent serum gives better blanching than antitoxin.

DR. TOOMEY.—Dr. Shaw has raised the question of practicability of the Schultz Charlton test. We agree that it is useless in any case that has a papulated or splotchy maculated rash and these usually are the very rashes about which we are in doubt. We agree, likewise, that there is no element of doubt in the typical scarlet fever case and the blanching test in those instances would be only of academic interest. Dr. Silverman stated that the test could not be used to rule out scarlet fever in an individual who has a typical rash. To this we agree, for we have seen hundreds of cases who do not blanch with the so-called commercial antitoxins. Some of these cases did blanch with convalescent serum. Incidentally, it is quite definite that one cannot sell scarlet fever antitoxins on the basis of blanching ability since most of the sera now sold have poor blanching qualities. If, however, blanching does occur with serum we do not see how one could ignore the possibilities such a reaction indicates.

II. Immunization

A. Active Immunization With Toxin Made From Streptococci.—All our practical experience has been with the use of Dick toxin as put out by the Squibb Company, the original licensees under the Dicks' patent.

In the question of immunization, two things have to be considered, the results of immunization and the possible dangers that might occur during its use.

For the past seven or eight years, one of us (J. A. T.) has been immunizing nurses who entered the local training school and over 953 students found to have positive Dick tests have been given the five immunizing injections recommended. Not one of the nurses had developed scarlet fever. Several entrants incompletely immunized have developed the disease.

The Dick test became negative in the immunized individual 10 days to 2 months after the last injection. Similar experiences are noted in the literature. Likewise, we have injected many children with similar results.

Are there any disadvantages in this program?

We have found some individuals so sensitive to the material that immunization had to be discontinued after the first or second injection. Reactions do occur with immunization programs. As a matter of fact, we have had but few nurses who have not had some reactions, the mildest of which were local inflammatory responses in 70 to 80 per cent of the group immunized. Approximately 10 to 15 per cent of these individuals were sick enough to be confined to bed from one to three days. Because of these reactions, we do not believe that the adults should ever be injected without an initial determination of their sensitivity; i.e., unless they have a positive Dick test.

Some say that adults may have reactions but that children do not, and that, therefore, injections should be made early, possibly between 2 and 5 years of age and made without even doing a Dick test. We cannot agree with the seemingly general opinion that children do not have reactions since we have had local and general reactions quite as severe as those seen in adults; therefore, we feel that children too, should have a determination of susceptibility before injections are given.

The local reactions referred to may be anything from a simple well demarcated lesion to an angry inflammatory response that involves the whole arm. The general reactions are those that occur with clinical attacks of scarlet fever, i.e., vomiting, severe prostration, high temperature, arthritis, adenitis, and often enough, a rash.

Because of these reactions, we feel that the method should be utilized to immunize actively only those individuals who may experience massive exposure in their work; i.e., nurses and doctors working in contagious hospitals or children in institutions. We do not feel that the procedure should be adopted as a public health measure since further work will have to be done to create a more innocuous product. One must remember also that active immunization does not necessarily give bacterial immunity, so that a person who either has had scarlet fever or has been actively immunized against scarlet fever may very well develop subsequent septic throats due to the scarlet fever organisms. We must understand that what we get when we inject five doses in the active immunization program is protection only from the subsequent formation of a rash in case the individual gets infected with one of the organisms that causes the disease.

DR. H. L. DWYER, KANSAS CITY, MISSOURI.—I wonder if on account of the reaction, the fourth or fifth doses might not be split. Most of my reactions occurred about the fourth and fifth dose. If we could cut these down, it might be that the number and severity of reactions might be decreased.

DR. SILVERMAN.—The number and type of reactions might be decreased but the number of injections would be increased from five to seven or ten. It would not be an easy problem to sell such a program to the public.

DR. FRANK LEECH, WASHINGTON, D. C.—My clinical experience with inoculation of nurses in our hospital at Washington has been equally as good as Dr. Toomey's at Cleveland. My results have been as fortunate as his since we have definitely cut down the number of scarlet fever cases that have occurred in our

nursing group. We, as did the speaker, experienced frequent reactions among the nurses, but I feel that it is a good measure to give to people who are intimately exposed to the disease. So far, I have had no case of scarlet fever occur among the inoculated internes and nurses. Though I feel it is a good measure in hospital work, I doubt its value as a public health measure.

DR. L. F. BENDER, PHILADELPHIA, PA.—We have been told that there are no reactions in children following injection of this material, but I have had severe reactions in my own family after active immunization injections. If other children have as severe reactions as my own, I would hesitate to utilize active immunization indiscriminately.

DR. SHAW.—I do not believe that in the attack of the scarlet fever problem, Dick testing, active immunization with toxin or toxoid, passive protection with scarlet fever antitoxin, or the treatment of scarlet fever by application of antitoxin, one dares to carry too far the analogy between scarlet fever and diphtheria. All of these control measures are designed with respect to the rash producing toxin as though its action were exactly comparable to the soluble toxin of diphtheria. Such a conception completely ignores the invasive and highly pathogenic properties of the streptococcus of scarlet fever which are apart from its production of soluble toxin and which are utterly unlike the almost purely saprophytic nature of the diphtheria bacillus. Whatever is to be accomplished in scarlet fever, prevention and treatment is to be accomplished only by direct observation of these therapeutic agents and not by analogy to diphtheria.

This is particularly true in public health work. To proceed with a campaign of scarlet fever immunization on the basis of our present knowledge, advising it to the public because of the success encountered in diphtheria immunization, might easily lead to results which would discredit not only scarlet fever immunization but diphtheria immunization as well.

DR. WALL.—After a patient becomes Dick negative after active immunization, how long can we say that the immunity lasts?

DR. DWYER.—If all we get by active immunization is protection against the rash, then there would be no sense in injecting susceptible individuals and advocating active immunization.

DR. G. B. MCFARLAND, DALLAS, TEXAS.—Is it not possible that due to active immunization people might contract a disease of bacterial form without a rash? This would tend to increase the instance of scarlet fever and break down the efficacy of quarantine laws. In other words, are the benefits obtained from active immunization worth while?

DR. TOOMEY.—Though we may admit some of Dr. Shaw's objections, nevertheless, we cannot shut our eyes to the striking results obtained by active immunization. This has been the experience of Dr. Leech here and others who have reported in the literature. For a hospital such a protection is worth while even though the injected individuals get reactions. The number of doses and the reactions accompanying the same are such that the program will never become generally popular. We cannot but feel, however, that from a clinical standpoint the patient, while perhaps still susceptible to the streptococcus causing the disease and perhaps only protected against the rash of the disease by the process of active immunization would probably be better off if he merely contracted a streptococcic sore throat than he would be if he had a streptococcus sore throat and a rash as might be the case if he had not had active immunization.

DR. EARLE.—I still think that active immunization is of value in private homes as among nurses in institutions. We have found that over 75 per cent of our patients were still negative two and a half years after the last active immunization

inoculation. Perhaps many of the reactions that occur with active immunization might be due to the additional allergic factor recently described by Ando and his associates. I have been using a toxoid preparation since January 1st and find that we do not get the terrific reactions with this material, but I have no definite figures to report as yet.

DR. TOOMEY.—We have not had a great deal of experience with toxoid although we have injected Larson's sodium ricinoleated material in some of our nursing groups. We discontinued it when we found that in some instances even after 15 injections, the nurses still had positive Dick tests. We have had no experience with the new toxoid material described by Dr. Earle.

DR. EARLE.—In our institutional work, we have cared for over 11,000 children. We have had no epidemics of scarlet fever since we started immunization.

B. Passive Immunization.—The reports from the literature as to the prophylactic value of scarlet fever antitoxin vary widely. Because of the complexities of individual immunity, the epidemic character of the disease, the variability of the sera, differences of virulency in organisms and the lowness of the contagious index, it is almost impossible for us to see how any comparisons can be made from which definite conclusions can be drawn.

There are, however, many articles in the literature by authors who think that they have seen definite evidences of value in these prophylactic injections, but the conclusions of most of these men were based on experiences with institutional cases. Injections were given as epidemics broke out and the claim is made that epidemics were thus controlled.

The variability of contagiousness in orphan asylums or institutions is so apparent to any one in medical charge of such institutions that one may assume that any conclusions based on possible case spread in these places are worthless. In the experience of one of us in one institution, one case has occurred one year with no subsequent infections and the very next year, 17 cases followed the first case of the season.

Because so few people contract scarlet fever anyway unless directly and intimately exposed, because even then the contagious index is low, because of the fact that the therapeutic sera sensitize to horse serum and finally because we do not feel that there has been sufficient data of a conclusive nature to prove that there is protection established by the injection of scarlet fever antitoxin, we feel that the prophylactic use of scarlet fever antitoxin is not to be recommended. If passive immunization has to be employed, we feel it should be with convalescent scarlet fever serum.

C. Conclusion.—We think that active immunization is a valuable procedure but is limited in its general application because of the marked untoward symptoms that accompany the procedure. Because of this, we feel that it should be used only to immunize individuals who are intimately exposed to the disease. We believe that passive immunization with the use of scarlet fever antitoxins in susceptibles is a procedure not yet justified either from the evidence in the literature or from our own experience and finally it is not justified since it may sensitize individuals to horse serum.

III. Treatment

A. Therapeutic Use of Scarlet Fever Antitoxins.—On another occasion, one of us* reviewed over 125 references on this subject. In this review, it is stated that the contributions in the literature could be divided into four classes.

*Toomey, John A.: Treatment and Prevention of Scarlet Fever by Specific Antitoxins and Serums, J. A. M. A. 91: 1599, 1928.

1. Original articles describing cases treated by antitoxins in great detail with results and proper controls.

2. Cumulative articles without details which merely draw general conclusions and which are impossible to analyze.

3. Articles whose authors used their control cases from other epidemics or patients treated with convalescent scarlet fever serum or other therapy.

4. Articles whose authors do not have controls.

At that time, reports of treatment to 4,611 patients had appeared in the literature.

The authors who favored the use of scarlet fever antitoxin stated that the rash disappeared earlier, the temperature came down more quickly, the patient was less toxic, got better sooner, and the rate of complications was lessened.

Some authors have said that the death rate was lowered. One may easily dispose of such claims since it would be inconceivable how any therapy would lower the death rate any further than we now experience. Curiously, the death rate of most metropolitan centers have been less than the rate quoted by one author who claims that his mortality was decreased. We cannot see how, in the light of vital statistic records, any one can seriously think that the mortality rate has been in any way affected.

As far as the disappearance of the rash is concerned, we can only state that the evidence obtained either from the literature or from our experience is not conclusive. The chief fault in reported articles lies in the fact that we are not told the length of time the patient had the rash before antitoxin was given. Usually the length of time between antitoxin administration and the disappearance of the rash is the time figured. One could not then conclude from a study of the literature, but one could admit for the sake of argument, that the rash disappears slightly more quickly in the treated than in the untreated case.

It is stated that the temperature comes down more quickly if antitoxin is used. We feel that the mass of printed evidence and our own experience does not seem to favor this conclusion. Silverman feels, however, that the temperature may be lowered more quickly.

As far as the toxicity is concerned, the literature is not very definite since most articles contain no reference to the length of time the patients were ill before receiving antitoxin. Nevertheless, we could agree with the general conclusion that the duration of toxicity is slightly less in the treated group; but this difference is anything but conclusive.

Some authors have had but few complications in their treated series in marked contrast to increases experienced in untreated cases. Reading the literature is confusing since the data present often do not substantiate the authors' conclusions. In the reported experience of one of us, there actually was an increase in the complication rate of the treated cases. Though this point had not been proved to our satisfaction, we might grant for the sake of argument that there may be a slight decrease in the complication rate following the use of antitoxins.

Are there any disadvantages that follow antitoxin injections? If so, might not these outweigh even the questionable advantages enumerated above? It is common opinion and it is our experience likewise that there are more reactions after the injection of scarlet fever antitoxin than there are after injections of any other type of serum. Just why this is so, we do not know.

The literature reports a very high percentage of serum sickness reactions in which the patients have had every type of response—early thermic, thermic with urticaria and edema, late urticaria and anaphylactoid phenomena. Most of the patients treated by us were far more ill from the serum sickness than they were from the scarlet fever.

Admitting all of the advantages various individuals claim, we still feel that there is no reason for injecting every patient with serum. The evidence obtained from the literature does not warrant concluding that scarlet fever antitoxin has proved of "inestimable value" in the treatment of this disease and we would not recommend it in the ordinary mild or moderately severely ill case. If given at all, it would be only to the strictly toxic patient in the hope that it might do some good. One of us would even object to its use here since he feels that convalescent scarlet fever serum, a homologous serum, accomplishes more beneficial results.

B. Conclusion.—From a practical standpoint, we would hesitate to recommend the use of scarlet fever antitoxins except in the occasional case and only when the patient has a strictly toxic type of the disease.

DR. JOSEPH YAMPOLSKY, ATLANTA, GEORGIA.—I would object to the use of scarlet fever antitoxin in prophylaxis because of the severe reactions that I have had in my practice. There reactions are not portrayed in the proper light to the private practitioner. They are much more severe than we would surmise from a cursory glance at the literature.

DR. SHAW.—I would heartily agree with the observer and say that if he was going to do any passive immunization, he should use convalescent serum.

DR. EARLE.—Might it not be a good idea in exposed susceptibles to begin giving skin test doses of toxin, in other words, give them active immunization?

DR. TOOMEY.—We feel that there is no good evidence on this point. One might reason that an individual who had been given injections in the incubation period and was about to get the disease might have the disease aggravated. Personally, we have had no experience along this line.

DR. SILVERMAN.—I also feel that active immunization of exposed susceptibles might aggravate scarlet fever if the disease were to develop shortly.

DR. YAMPOLSKY.—I formerly used scarlet fever antitoxin, but because of the severe reactions I obtained I now only use it in selected cases and give it in very small doses. My difficulty is the ability to differentiate toxic cases early. I find that reactions are very severe if you give large doses.

DR. SHAW.—I feel that the fall in temperature reaction which occur naturally in such a disease as scarlet fever is very apt to be attributed to the antitoxin. I doubt whether or not antitoxin is of any value. I feel with one of the speakers that in a severe case, convalescent scarlet fever serum would probably be of more use.

DR. J. B. STONE, RICHMOND, VIRGINIA.—I wonder when convalescent scarlet fever serum is not available whether the serum of an individual with a negative Dick test might be utilized as a therapeutic agent.

DR. TOOMEY.—I do not know of any articles bearing on this point.

DR. SILVERMAN.—We do not know the immunity titer of a patient's serum and so we cannot tell the antitoxic value of a patient's serum in a quantitative way. I feel that patients with any hint of allergy should have convalescent serum.

DR. EARLE.—Do you have to type the blood for convalescent serum?

DR. TOOMEY.—Not if the blood is to be given intramuscularly as it usually is or intraperitoneally. If given intravenously, typing will have to be done.

DR. EARLE.—I gave antitoxin in curative doses to two children in one family. In one instance, I had no reaction, good results and quick recovery. In the other child, we had no evident results. I feel that antitoxin has a limited use in some cases.

DR. SILVERMAN.—The fact that those individuals who get good blanching tests have good results with the serum we have used makes me feel that the fault lies with the antitoxin. We use the material supplied by the New York State Board of Health.

DR. DWYER.—I feel that adult's serum is in a way comparable to convalescent serum in that it has some relative value and might be used.

DR. TOOMEY.—We did not use any of the New York State Board of Health's antitoxin in our blanching tests. From the published literature and from our experience with men who have used their material, there is no question but what the New York State Board of Health manufactures a superior brand of anti-streptococcic antitoxin.

DR. McFARLAND.—I wonder whether some one could tell us what the criterion in scarlet fever would be for diagnosis of scarlet fever in doubtful cases. Recently, we have had many cases with a very fine rash that have been usually diagnosed scarlet fever. These cases had some fine pecling.

DR. TOOMEY.—I would say that the individuals had scarlet fever. They had a rash, though scanty, desquamation, though fine, and a sore throat, though mild.

DR. STONE.—I wonder whether the chairman could tell us anything about the nature of the rash after tonsillectomy.

DR. TOOMEY.—I do not know the mechanism. It might be that streptococci could secondarily infect this area or the protein absorption that occurs about the wound could cause this reaction.

DR. EARLE.—Do cases always have desquamation after scarlet fever?

DR. SILVERMAN.—I have seen many frank cases of scarlet fever that did not desquamate.

DR. J. B. STONE, RICHMOND, VIRGINIA.—What is the significance of throat cultures in this disease?

DR. SILVERMAN.—I think that the consensus of opinion is that we cannot utilize culture methods as a means for isolation or even as a criterion of discharge from this disease.

(The seminar on the above points was opened with Dr. Toomey presiding. In the absence of Dr. Hoyne of Chicago, Dr. J. B. Stone of Richmond acted as secretary.)

Academy News

Dr. Roger H. Dennett of New York City has been appointed Chairman of the Committee on Nursing Education in Pediatrics to replace Dr. Ramsey who has resigned. Dr. Charles A. Fife of Philadelphia has accepted a position as a member of this same committee.

Dr. Herbert L. Moon of Seattle has been made Chairman of the State Committee of Washington, to succeed Dr. Jay I. Durand.

Dr. Stanley Nichols of Asbury Park has succeeded Dr. Joseph H. Marcus as State Chairman in New Jersey.

News and Notes

Professor N. I. Krasnogorsky has left for Russia to resume his duties as chief of the first Pediatric Clinic at Leningrad.

Following the meeting of the American Pediatric Society at Rochester, Minnesota, where he delivered an address as foreign guest of the Society, he visited several pediatric clinics and was at the University of Chicago for a period of two weeks. After this he lived at the Babies' and Children's Hospital in Cleveland from July 1 to October 10, where he continued with members of the hospital staff, particularly Dr. Charles Good, his research on Conditioned Reflexes.

Dr. H. J. Gerstenberger has returned from Europe. While there he attended the annual meeting of the Deutsche Gesellschaft für Kinderheilkunde held in Vienna from September 21-24, where he read a paper on "Hepatic Rickets."

Dr. Irving M. Snow of Buffalo, New York, died October 17, 1932.

New Legal Regulations for the Practice of Medicine in France

1. In order to practice medicine in France, it is compulsory to be a French citizen or citizen of a French Colony or Protectorate.

2. To hold a State Diploma of Doctor of Medicine

Art. 2. Owners of foreign diplomas, in order to obtain a French diploma must first prove their original degrees in order to obtain special exemption of study not exceeding three years.

Art. 4. Diplomas of Doctors of Medicine already practicing in France should all be verified and legalized within 3 months.

Art. 5. No State function can be fulfilled by a foreign doctor or a naturalized French subject of less than five years' standing.

Art. 6. French naturalization will not be demanded from those already in practice and in possession of a French diploma or who have already entered on a course of study in a French faculty for the said diploma.

Infant Mortality

Mortality among dependent children of under one year in the Department of the Seine has decreased from 36 per cent to 9.95 per cent.

Four years ago, the Director of the "Assistance Publique" showed to the French Academy of Medicine statistics of the mortality among dependent infants brought up in welfare centers.

Not long ago a special Center for adapting the infant's diet to cow's milk was opened near Paris. With the introduction of welfare centers, mortality which was 36 per cent in 1920, was reduced to 15.77 per cent in 1922; 16.94 per cent in 1923; 14.44 per cent in 1924; 15.14 per cent in 1925; 16.82 per cent in 1926.

Following the opening of a center for adapting cow's milk to babies' requirements in 1927, mortality has fallen to 9.42 per cent in 1928; 7.43 per cent in 1929; 8.14 per cent in 1930; 5.95 per cent in 1931.

These figures concern dependent children of under one year, including abnormal and subnormal infants.

BIRTH RATE AND INFANT MORTALITY IN FRANCE FROM 1926 TO 1930

BIRTH RATE PER 1000 INHABITANTS

	DEPARTEMENTS	BEST
1.	Pas-de-Calais	25,13
2.	Basses-Pyrénées	
3.	Moselle	23,64
4.	Meurthe-et-Moselle	23,42
5.	Morbihan	22,54
6.	Calvados	22,27
7.	Manche	21,98
8.	Seine-Inférieure	21,67
9.	Aisne	21,62
10.	Côtes-du-Nord	21,56
11.	Mayenne	21,22
12.	Ille-et-Vilaine	21,21
13.	Finistère	21,04
14.	Doubs	20,97
15.	Vendée	20,77
16.	Nord	20,51
17.	Meuse	20,39
18.	Sarthe	20,36
19.	Marne	20,30
20.	Eure	20,22
21.	Bas-Rhin	20,11
22.	Vosges	20,04
23.	Orne	20,03
24.	Oise	19,96
25.	Belfort	19,88
26.	Ardennes	19,86
27.	Haut-Rhin	19,74
28.	Somme	19,53
29.	Eure-et-Loir	19,44
30.	Savoie	19,17
31.	Deux-Sèvres	18,82
32.	Haute-Marne	18,79
33.	Aube	18,71
34.	Haute-Savoie	18,62
35.	Loire-Inférieure	18,61
36.	Jura	18,49
37.	Maine-et-Loire	18,35
38.	Cantal	18,20
39.	Aveyron	18,03
40.	Hautes-Alpes	17,96
41.	Haute-Saône	17,90
42.	Charente-Inférieure	17,79
43.	Seine-et-Marne	17,72
44.	Ardèche	17,63
45.	Indre-et-Loire	17,49

	DEPARTEMENTS	WORST
1.	Corse	14,09
2.	Creuse	14,27
3.	Ariège	14,31
4.	Lot	14,40
5.	Gers	14,54
6.	Pyrénées-Orientales	14,59
7.	Cher	14,69
8.	Allier	14,73
9.	Var	14,85
10.	Nièvre	14,94
11.	Hérault	15,14
12.	Gard	15,15
13.	Puy-de-Dôme	15,40
14.	Basses-Alpes	15,42
15.	Haute-Garonne	15,43
16.	Lot-et-Garonne	15,53
17.	Tarn-et-Garonne	15,69
18.	Aude	15,84
19.	Corrèze	15,88
20.	Yonne	15,90
21.	Haute-Vienne	16,00
22.	Drôme	16,07
	Hautes-Pyrénées	16,07
24.	Indre	16,12
25.	Gironde	16,13
26.	Alpes-Maritimes	16,14
	Landes	16,14
28.	Tarn	16,37
29.	Rhône	16,42
30.	Bouches-du-Rhône	16,48
31.	Seine-et-Oise	16,52
32.	Seine	16,59
33.	Vaucluse	16,64
34.	Seine	16,59
	Dordogne	16,76
36.	Saône-et-Loire	17,03
37.	Loiret	17,06
38.	Ain	17,12
39.	Haute-Loire	17,16
40.	Charente	17,20
41.	Isère	17,23
42.	Côte-d'Or	17,25
43.	Lozère	17,31
44.	Vienne	17,38
45.	Loire-et-Cher	17,46

INFANT MORTALITY 1926 TO 1930 PER 1000 INHABITANTS

DEPARTEMENTS	BEST	DEPARTEMENTS	WORST
1. Landes	53,52	1. Calvados	114,13
2. Vendée	54,11	2. Haute-Loire	110,77
3. Creuse	57,16	3. Seine-Inférieure	110,09
4. Vienne	60,13	4. Pas-de-Calais	108,19
5. Cher	60,27	5. Seine-et-Oise	107,64
6. Indre	61,59	6. Cantal	106,73
7. Deux-Sèvres	64,922	7. Ardèche	106,58
8. Haute-Vienne	67,30	8. Sarthe	106,21
9. Nièvre	67,84	9. Seine-et-Marne	105,47
10. Haut-Rhin	68,41	10. Moselle	104,38
11. Allier	68,79	11. Meurthe-et-Moselle	103,56
12. Loire-Inférieure	69,89	12. Oise	102,01
13. Gers	69,96	13. Aveyron	100.80
14. Loire-et-Cher	70,59	14. Somme	100,55
15. Charente-Inférieure	71,06	15. Bouches-du-Rhône	100,46
16. Loiret	72,24	16. Eure	98,82
17. Maine-et-Loire	72,51	17. Aube	98,60
18. Puy-de-Dôme	74,19	18. Rhône	98,48
19. Indre-et-Loire	74,28	19. Corse	98,42
20. Jura	74,29	20. Orne	97,37
21. Dordogne	74,78	21. Côtes-du-Nord	96,79
22. Côte-d'Or	74,79	22. Isère	96,59
23. Lot-et-Garonne	75,03	23. Vosges	96,43
24. Finistère	75,46	24. Loire	96,32
25. Haute-Garonne	75,50	25. Seine	96,27
26. Belfort	75,60	26. Hautes-Alpes	95,96
27. Charente	76,40	27. Tarn-et-Garonne	95,84
28. Saône-et-Loire	76,81	28. Eure-et-Loir	95,81
29. Ain	77,24	29. Morbihan	94,47
Ariège	77,40	30. Ille-et-Vilaine	94,01
30. Tarn	78,	31. Nord	93,23
31. Hérault	78,71	32. Savoie	93,03
32. Mayenne	79,11	33. Meuse	90,96
33. Haute-Savoie	79,22	34. Lot	90,57
34. Vaucluse	79,58	35. Basses-Alpes	89,78
35. Bas-Rhin	79,77	36. Marne	89,68
36. Basses-Pyrénées	79,88	37. Alpes-Maritimes	89,33
37. Gironde	80,97	38. Aisne	87,93
38. Ardennes	81,33	39. Var	87,84
39. Drôme	81,66	40. Pyrénées-Orientales	87,30
40. Lozère	82,85	41. Haute-Marne	86,73
41. Haute-Saône	83,37	42. Hautes-Pyrénées	86,33
42. Gard	83,97	43. Yonne	85,57
43. Aude	84,37	44. Manche	85,52
44. Corrèze	84,52	45. Doubs	84,67

Professor Marfan states that at the Hospice des Enfants Assistes mortality was 50 per cent in 1920, 14 per cent in 1927 and has been reduced to 7 per cent in 1931. This has been the result of scientific work which has resulted in the opening of the above-mentioned Center.

Infant Mortality and Birthrate in Europe.—Dr. Cauchois of Rouen (Seine Inferieure) has given in a local paper the classification of towns with the lowest and highest infant mortality and birthrate in Europe from 1926 to 1929.

THE HIGHEST

TOWN	MORTALITY PER 1000	BIRTHRATE PER 1000 INHABITANTS
Lodz	170	19.47
Rouen	158	22.62
Leningrad	148	42.30
Seville	138	28.60
Moscow	138	26.20
Warsaw	135	19.50
Padua	123	21.19
Grenada	121	24.4
Saragossa	121	26.60
Moravoska	121	13.42
Madrid	120	25.52
Cracovia	120	22.7
Venice	119	21.85
Naples	117	24.00
Malaga	116	33.47
Murcia	115	31.35
Bratislava	115	21.05

THE LOWEST

Berne	34	13.25
Oslo	39	11.25
Zurich	40	13.43
Amsterdam	41	18.42
The Hague	42	17.42
Rotterdam	48	20.42
Basle	48	12.00
Stockholm	50	11.00
Stuttgart	58	12.27
Frankfort	58	11.8
Copenhagen	60	16.32
Bristol	71	16.65
Hamburg	76	12.60
Antwerp	77	16.35
Brussels	78	12.00
Düsseldorf	79	14.75

The principal difference in these statistics appears on the one hand, to be geographical, mortality being less in northern countries and increasing toward southern latitudes; and on the other hand, ethnical. A large factor is squalidness, alcoholism and tuberculosis.

DR. G. ILLINGWORTH HELIE,
Paris, France.

Book Reviews

Appraisement of the Child. Report of the Committee on Growth and Development, White House Conference on Child Health and Protection. The Century Company, New York, 1932, p. 344.

The volume on *Appraisement of the Child* is composed of two parts; the first being the report of a subcommittee dealing with Mental Status; and the second, the report of one dealing with Physical Status.

Appraisal of Mental Status.—As mental growth had not received separate consideration in other volumes, the report on Mental Status opens with a general description of mental phenomena, and of the course of mental growth. It also contains special chapters on the development of motor skills, language, and behavior, and on mentally superior and inferior children. It deals with the effects of nutrition and organic disturbances, as well as of training, education, and socio-economic conditions upon the course of mental growth. It also considers the possibilities of protecting mental growth through supervision of training and care. The necessity for including this material makes the section on Mental Status appear to dominate the report and to relegate Physical Status to the background. The chapters actually dealing with the appraisement of mental status, however, are well balanced with those dealing with physical status. They present a careful scientific analysis of the possibilities inherent in our present techniques for appraising intelligence at the various age levels; and deal in the same way with tests of special aptitudes, of motor skills, and of personality.

The first chapter of the Mental Status section entitled "Mental Growth in Infancy and Childhood" will prove of special interest and value to anyone concerned with the broad problem of protecting the health of children, for it defines the place of mental in relation to total health, and indicates the part which the observation and control of behavior may be expected to take in this field. Some of the practical conclusions which may be culled from this chapter are the following:

> Mental development is very orderly and predictable. "For every age group and for every individual there are ascertainable *norms* of behavior which must enter into an estimate of mental and physical health." The sequence and rate of progress, however, are of far greater importance than status at any moment; and, therefore, the accuracy and value of developmental diagnoses are greatly enhanced by periodic follow-up examinations.
>
> The diagnosis of behavior development is more than an estimate of general intelligence, and a single simplified mental rating becomes misleading, unless interpreted in terms of the child's total individuality and his previous developmental career.
>
> Departures from normal development, as well as illnesses, express themselves in more or less distinctive behavior signs; and, hence, developmental and health supervision must include consideration of mental progress and behavior disorders.
>
> The periods of infancy and preschool childhood are of peculiar medical significance, and afford a favorable time for preventive mental hygiene. "The behavior signs of significant deviations (from orderly development) must be sought and found early."

A summary of the content of the various chapters which follow cannot be attempted, as they are themselves very brief summaries of the mass of existing knowledge relating to the different topics. Among the more practical chapters of interest to layworkers, but of equal value for the medical profession, are those entitled "Behavior in Normal Childhood and Adolescence" and "Training in Habits." Owing to the great interest which is attached to the problem of testing intelligence the summary presented at the end of the chapter dealing with this subject is quoted here in full:

"Intelligence tests have proved themselves a useful means of correcting and supplementing the more subjective methods of estimating human abilities. They have thrown important light on the rate of mental growth and on the problems of individual differences generally. They are indispensable tools in the study of exceptional children, and have considerable value as a partial basis for educational and vocational guidance. Their shortcomings, which have been pointed out by their authors as well as by their critics, should not be lost sight of. It is not claimed that they test every aspect of mental ability, that they are an infallible measure of original capacity, or that the scores they yield are equal units in the sense that units of physical measurement are equal. It is recognized that apart from general intelligence there are special abilities and various combinations of personality traits which contribute very significantly to any individual's social value. Some of the more important tests, both of general intelligence and of special aptitude, have been briefly described, but there has been no attempt to include in these descriptions all of the useful tests that have been devised. In considering the use of any test, it is important to take account of all the available evidence as to what the test measures, and how reliably it measures what it purports to measure. It is especially important not to be misled by the apparent definiteness suggested by the numerical scores of tests."

Appraisal of Physical Status.—The Section on Physical Status limits itself strictly to subjects which are related to appraisement, because the whole field of physical development has been reviewed in previous volumes. The report starts with a consideration of norms of physical measurement and their use in clinical appraisement. The difficulties with norms are emphasized, difficulties both with the accuracy and comparability of individual measurements, but of greater importance, difficulties in the interpretation of variations from the standard. It goes so far as to question the possibility of ever using measurements as tests of the physical fitness of the individual, for any individual must fall outside the range of the physically fit group before he can be differentiated from that group. This conclusion obviously refers only to the use of measurements taken at one time and denoting status. It seems unfortunate to the reviewer that the greater value of comparative measurements of the same individual, taken at consecutive times, is not brought out in this connection. There is no doubt that repeated measurements can be used effectively as tests of progress and rate of growth, and that these are intimately related to physical fitness. Weight alone has proved of real value to clinicians in this connection. The report itself states in a later chapter: "The weight of a child should be fitted into his own weight curve, not that of other children. Change of weight and rate of gain in the individual himself are the really significant measures of his progress."

The measurement of quantity and appraisal of quality of soft tissues, and their relation to nutritional status are briefly considered in another chapter. Emphasis is given to the fact that height-weight ratios are more largely influenced by differences in body build or skeletal structure than by differences in quantity of soft tissues. Hence, these are not suitable for verdicts as to the adequacy of the individual's diet or nutritional processes. Nor did the Committee feel that it would be practical to build up an index of nutrition based upon more numerous skeletal

measures, owing to the difficulties involved in the use of such mathematical representations. Furthermore, the disagreement existing between different observers in their subjective evaluations of clinical signs, supposedly relating to nutrition, renders the need for more objective measurements of these items very urgent. Hope is entertained that the use of calipers and tape may be developed into serviceable means of recording both quality and quantity of subcutaneous tissue and muscle, but the limitations of the present technics for their use are recognized.

One chapter deals with the measurement of socio-economic factors as they influence the school child. It considers the various kinds of influences which combine to make up the socio-economic environment, the methods of measuring each of these, and their relative reliability and importance.

An interesting chapter follows on the uses to which the roentgenogram may be put, as a measure of the development of the skeleton, and the quality of the bony tissues. The technic to be adopted, when used for these purposes, is described.

A chapter on the vital capacity of the lungs as related to physical status clearly presents the limitations of this procedure, and considers it entirely impracticable for use with children under eight years of age. It does, however, conclude by recommending its use with older children "as a routine under conditions that will insure accuracy." If this advice were followed, it would constitute a very real departure in health practice; but the conclusion hardly seems to the reviewer to be justified by the evidence of its value presented in the rest of the chapter.

The volume ends with a summary of the practical features of physical appraisement, and should be of particular interest and usefulness to physicians and others who have to deal with questions of child health. It reviews the important factors to be considered in the appraisement: first, of populations of children as in countries, local areas, and the like; then, of groups of children as in classes at school; and last, of the individual child. The narrow limitations of the first kind of appraisal are made obvious. The restricted, though useful, functions which school examinations serve are clearly indicated. These functions are listed as

(1) Educational
(2) Protection of one child from disease in another
(3) Assuring the fitness of a given child for the routine to be imposed upon him
(4) The finding of defects and early recognition of disease, and calling the attention of family and family physician to their presence.

The report suggests that it is in connection with the last of these functions that school health services have most commonly led to unwarranted activities. The difficulty is that most of the items in a physical examination taken singly serve only as indicators, and cannot be accepted as criteria upon which diagnoses are to be made or recommendations for procedure developed. These usually require further careful individual study, of a nature rarely provided in school programs, before their real significance can be ascertained. It is suggested that "unusual findings detected in school examinations should be reported as indications of possible defects, requiring further study and not with appended diagnoses or therapeutic recommendations."

The individual health appraisal is presented as the only kind that will adequately determine the needs and protect the health of a given child, because he has an individuality which modifies his needs and his reactions to environment. The extensiveness, as well as the intensiveness, necessary for such appraisals is considered at some length. The greater value of periodic, or serial, examinations over a single examination of status is particularly emphasized, just as it was in connection with mental appraisal.

If this volume is widely distributed among those responsible for the examination of children, and is given thoughtful attention by them, the value of appraisements in the future will unquestionably be greatly enhanced.

HAROLD J. STUART, M.D.

Comments

APPRAISEMENT OF THE CHILD'' which is reviewed in this issue is the fourth of a series of reports prepared by the Committee on Growth and Development of the White House Conference under the chairmanship of Kenneth D. Blackfan, M.D. It is probably safe to say that no publication has been more sorely needed than this volume dealing with the objectives and methods of health appraisement in childhood. Periodic physical examinations of normal children have been carried out in connection with school health programs, and as integral parts of various health supervisory services, in rapidly expanding numbers. There has been in recent years an increasing public demand for more effective child health protection, and various programs directed toward meeting this demand have been instituted. Most of these include routine physical examinations, and physicians have been called upon to participate to the extent at least of carrying out these examinations.

Those who have been responsible for administering health programs have paid surprisingly little attention to the content and quality of the physical examinations provided. There has also been comparatively little thought given to the possibilities inherent in such examinations, and their inevitable limitations. The brief exposure of a partially clothed child to a man who possesses a medical degree has seemed to satisfy the requirements in many instances. Medical schools have also neglected to provide adequate training in the technic of preventive health examinations, or to afford a sufficient background of knowledge relating to the growth and development of normal children to fit students to interpret their findings when making health appraisals. The physicians, who have been drafted into this service, have applied the technic which they were taught was appropriate for the examination of sick children, and have commonly confined their attention to the finding of manifest disease. Largely owing to this limited viewpoint, the health examination has proved dull and unprofitable to them. Even in the biological sciences, students have, with few notable exceptions, failed to devote the attention which the subject obviously warrants to evolving useful objective criteria of health and normal development in growing children.

It is high time that both laymen and physicians appreciate the limitations of our present knowledge and technics, and realize that because of these limitations the appraisement of the normal child is difficult. The volume under consideration presents these difficulties and makes it clear that special study and training, as well as attentive and thorough effort in application, are required if health examinations are to be effective. Furthermore, it constantly calls attention to the need for more fundamental studies to provide the knowledge necessary to make appraisements more reliable. It particularly warns against the too ready acceptance of any of the items customarily used in examinations as *criteria* of health. The following quotation gives some of the requirements which must be met before any item can be accepted as thoroughly satisfactory, and indicates how far most of those in common use fall short of meeting acceptable standards: "Reliable appraisement of physical status demands that the items on which dependence is placed shall be valid measures of relative physical fitness; that the measurements be as objective as possible; that the results obtained shall be capable of repetition in identical circumstances; that the nature of the measurements be such that fluctuation from the standard is of proved significance; and that the standard, or norm, shall be applicable to the individual, not just an average from a physically heterogeneous group."

While reading certain chapters, one wonders whether the attempt to assess health is not futile, and whether the whole movement to provide periodic health examinations for normal children is not fundamentally unsound. This destructive criticism is, however, necessary before an adequate conception of what is essential, and what is possible in health service, can be constructed. Having provided this groundwork, the report goes on to show that by comprehensive and repeated assessments of the individual, and by the use of many items as indicators rather than criteria of health, conclusions of great practical usefulness can be drawn from health appraisals. Rather than discounting their importance, the report, taken as a whole, opens up new vistas as to their possibilities; and leaves the reader with the firm conviction that we have only started on a movement to protect the health of children through periodic health assessment, and that this procedure holds great promise for the future.

THIS issue completes Volume I of THE JOURNAL OF PEDIATRICS. The Editorial Board has endeavored to hold to its original plan of covering the field of childhood as related to pediatrics and it proposes to continue this plan. The encouragement received has been most gratifying. Despite the period of economic depression in which the journal was started, the subscription list has grown and is continuing to grow beyond expectation. Its value as an advertising medium has been recognized. These two factors are essential from a business standpoint. Whether we like it or not the type and character of the original communications expresses current American pediatric thought and work. In other words, the quality and character of the Journal depends upon the contributions received for publication.

We have not received the criticism we have hoped and looked for. We have been disappointed in not receiving material for this column of "Comment." Perhaps our readers have been too charitable and have felt the Editors would improve with age. We recognize many of our shortcomings, but we have no apology to make. THE JOURNAL OF PEDIATRICS belongs to the pediatricians of America. As the official organ of the Academy of Pediatrics it is the responsibility of every member and not simply a responsibility of those who were selected for the Editorial Board. If it is to grow and thrive and improve it requires your interest and help.

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